BRAIN.

PART IV., 1903.

Original Articles and Clinical Cases.

CASE OF PROGRESSIVE MUSCULAR ATROPHY OF SPINAL ORIGIN IN A GIRL AGED 11 YEARS; WITH AUTOPSY.

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AND

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THE following case is an example of a progressive muscular atrophy of spinal origin occurring in a child. The case, both from its clinical and pathological aspect, is unusual and forms a member of a group of cases which, although they may be classed together as muscular atrophy, yet in their onset, their course, and also in their morbid anatomy and pathology, differ considerably from one another.

We have been unable to find any record of a case similar to the one here described, and although for the present this case is grouped with those described by Werdnig and Hoffmann, yet it is fully recognised that it does not either clinically or pathologically exactly coincide with the description of the disease given by these authors. On the other hand, it seems advisable for the present to keep one large group of "progressive muscular atrophy of spinal origin in children," and at some later period, when pathological

observation is more extended, to arrange the cases into groups based on more accurate knowledge.

Clinical History.

M. L., 11 years. Twelve months weakness of hands. Seven months unable to button clothes. Four months weakness of legs. Two months adenoids removed, and much worse since that operation. Fourteen days pain in back and increased difficulty in swallowing. Intelligent, deaf, some atrophy of tongue, palate moves well on phonation, weakness of neck, respiration thoracic, upper intercostals only in use. Claw hand, wrist drop, weakness of upper arm and shoulder muscles. Can walk but only feebly, knee jerks brisk, plantars flexor, sensation natural to all forms. Electrical reactions R.D. in some muscles, in others only diminution of irritability. Sudden death from respiratory failure six months later.

M. L., aged 11 years, was admitted into East London Hospital for Children, Shadwell, on November 25, 1901, under the care of Dr. Fletcher. The following is an abstract from the ward notes:—

The *history of present illness* was as follows: Weakness in legs and weariness after any exertion were the first symptoms noticed. She complained of pain in the chest, and had a cough, but this ceased three months ago. About a year ago the hands were noticed to be tremulous, and she used to drop things. She had not been able to button clothes for seven or eight months. She could walk fairly well four months ago, but the legs had got much weaker lately. Two months ago she had had adenoids removed, and since that operation she had been much worse, and had wasted very rapidly all over, and had become unable to hold a cup to feed herself. She had complained of great pain in the back for a fortnight, and been in bed since then. She took her food very badly, and seemed to have difficulty in swallowing. There had been vomiting; the bowels had been constipated; there had been no difficulty with micturition. She had been deaf for two years, and worse since the operation on the throat.

Previous history.—She had always been delicate, but there had been no acute illness. There was no evidence of congenital syphilis.

Family history.—Mother "languid." Father died about five years ago, cause unknown. There were three other children

living, boys, who were said to be delicate; four dead—one pneumonia, three bronchitis; no miscarriages. Instruments were used at the birth of this child. Grandmother had a sister and a cousin in an asylum. Mother's sister is in an asylum; a cousin had fits.

On admission the girl was emaciated, but the face was not wasted as much as the rest of the body. She seemed intelligent, but it was difficult to make her understand as she was very deaf. On the left side she could hear a watch at about one and a half inches; on the right side could not hear a watch at all. She would answer questions readily when she heard them; her voice was natural.

Special senses.—Sight, taste and smell good.

Eyes.—Pupils were of medium size, equal, and reacted to light and on accommodation. Ocular movements were normal. The discs were pale; no alteration was observed in vessels; the discs showed physiological cupping. No weakness or paralysis of face was made out. The tongue was well protruded in the middle line; it was coated, was tremulous, and the edges appeared to be thinned. The soft palate moved well and equally on phonation and respiration. She had some difficulty in swallowing, especially solids, and could only swallow slowly.

Trunk muscles.—Respiration was entirely thoracic; only the accessory muscles of respiration and upper intercostals were in use. The thorax was flat in the antero-posterior direction, and long in a vertical direction.

The lower ribs seemed closer together than normal, and expanded very little during respiration; when an attempt was made to sit up with the hands on the chest the recti abdominis could be felt to contract, but were weak; the umbilicus tended to move down. The chest and abdomen were much wasted. The girl could sit up with the aid of her arms. The head could only be held up for a short time, and tended to fall forwards when sitting up. The muscles at back of neck seemed much weaker than others. The muscles of the back were extremely wasted, and this wasting was more marked in the lower dorsal and lumbar regions than higher up.

The upper part of the trapezius was present on both sides, but was wasted. The supra- and infra-spinati and the serratus magnus were all wasted, but not completely paralysed. The fibres of the clavicular part of the pectoralis major were present, but much wasted, the lower part was much less atrophied. The deltoid, triceps, and biceps were small and weak, the muscles of

the forearm were also wasted, the flexors more than the extensors; supination and pronation could be performed.

Flexion at the wrists was present but weak, there was wrist drop on both sides. Extension at the wrists was very feeble, but the hand could be extended a little on both sides. The hands were extremely wasted—the fingers being in the position of claw hand—there was much wasting of the interossei and thenar muscles, with much less-marked wasting of the hypothenar muscles. The first dorsal interosseus could be felt to be present on both sides. All the small muscles of the hands seemed to act except the opponens of the thumb; the tips of the thumb and little finger could not be brought together.

There was a fine tremor of the hands when held out (this appeared to be due to weakness). There was some incoördination of the hands, but this was not marked. Muscular sense as tested by weights was not good. The legs were thin and the muscles were generally wasted. All movements could be performed, and the relative strength of the legs was much greater than in the arms. There was no foot drop and no tenderness of muscles or nerve trunks. There was some stiffness when the patient attempted to flex the knees and ankles, but this was easily overcome and did not continue. The patient could walk by herself, but was weak and tended to fall to one side or the other. The gait was rather spastic in character. The knee jerks on admission were very exaggerated on both sides, and the depressed patella jerk was active. On the following day the knee jerks were still exaggerated, but not nearly so much as on first examination. The Achilles jerk was present on both sides. The plantar reflexes gave flexor responses on both sides. Abdominal and epigastric reflexes were not obtained.

Tactile sensation was good all over the trunk, limbs and face. There was no analgesia and no loss of sensation to heat and cold.

Spine.—There was some prominence of lower dorsal spines, but no tenderness. There was a slight lateral curvature in the dorso-lumbar region with convexity to the right.

The child had incontinence of urine on one occasion. There was no vomiting; the bowels were constipated.

Nothing abnormal could be detected on examination of the chest or abdomen. The pulse was 98, soft, regular. Urine 1030, acid, no albumen.

November 30.—The child breathed easily when awake—whilst during sleep the respirations were more rapid, and seemed

laboured. The accessory muscles of respiration and the intercostals alone were in use. The colour remained good. The pulse was usually about 100, though sometimes more rapid (120-140 per minute).

On December 10 the following note was made:—The lateral movements of the tongue against the cheek were weak, and there was considerable fibrillary tremor when first protruded. The power of swallowing solids was rather improved. The muscles of the chest wall had become more wasted during the past week, especially in the infraclavicular region. Respiration was still carried on chiefly by the accessory muscles, the abdominal muscles being motionless. On attempting to sit up the umbilicus moved in a downward direction, and the abdominal and epigastric reflexes were not obtained. The knee jerks were still active, and the plantar reflexes were flexor in type.

The child remained in much the same condition during the two months she was in hospital, her general condition improved and she was bright and merry and asked to get up. Respiration remained almost entirely thoracic, and the child still had some difficulty in swallowing. The atrophy of the tongue became more apparent. The knee jerks remained active, and the plantar reflexes gave flexor responses.

On January 28 the child was transferred to St. Bartholomew's Hospital under the care of Dr. Gee, to whom we are indebted for permission to publish the further history of the case.

The following additional notes were made:—

Electrical reactions.—Dr. Lewis Jones reported that "in the left hand there was reaction of degeneration in the abductor and opponens pollicis. In the interossei of that hand the reactions vary; to the coil they were not quite lost, but feeble in varying degrees; to the cells inclined to sluggishness. The right hand—reactions lost in thenar eminence, both in ulnar and in median muscles. First dorsal interosseus, decided R.D.; first palmar, probably similar; other interossei uncertain, shading into fairly good contractility to coil and to cells in hypothenar. The arm and forearm muscles and the thigh and leg muscles seemed to have good normal reactions—apparently contractility is augmented in all, but the patient is very timid and seems to feel the testing acutely." (Signed H. L. J.).

February 23.—There was very little change in her general condition, but what there was seemed to be towards a slight

improvement; she could walk well by herself, and felt no difficulty in breathing, though the diaphragm and lower intercostals did not act.

On March 1 she had an attack of dyspnœa with cough, she had slight bronchitis with a moderate rise of temperature. She died quite suddenly from respiratory failure on March 2.

The *post-mortem* examination was made by one of us (H. M. F.) twenty-six hours after death.

The external appearances were those mentioned in the clinical note.

The brain weighed 46 ozs., and appeared normal externally. No abnormality was found in the membranes, sinuses, or vessels.

The brain and spinal cord were removed and at once placed in a formalin solution.

The intercostal muscles were greatly atrophied and presented an almost membranous appearance in the lower intercostal spaces. The diaphragm was of extreme thinness, resembling a transparent membrane, and little muscular tissue could be detected therein. The lungs were congested, the bronchial tubes contained muco-pus, and the left lower lobe was nearly solid, probably due to hypostatic congestion. There was a patch of recent pleurisy on the left side of the diaphragm.

The other organs were natural. The following peripheral nerves were removed: the phrenics, the median, and ulnar.

Pathological Examination.

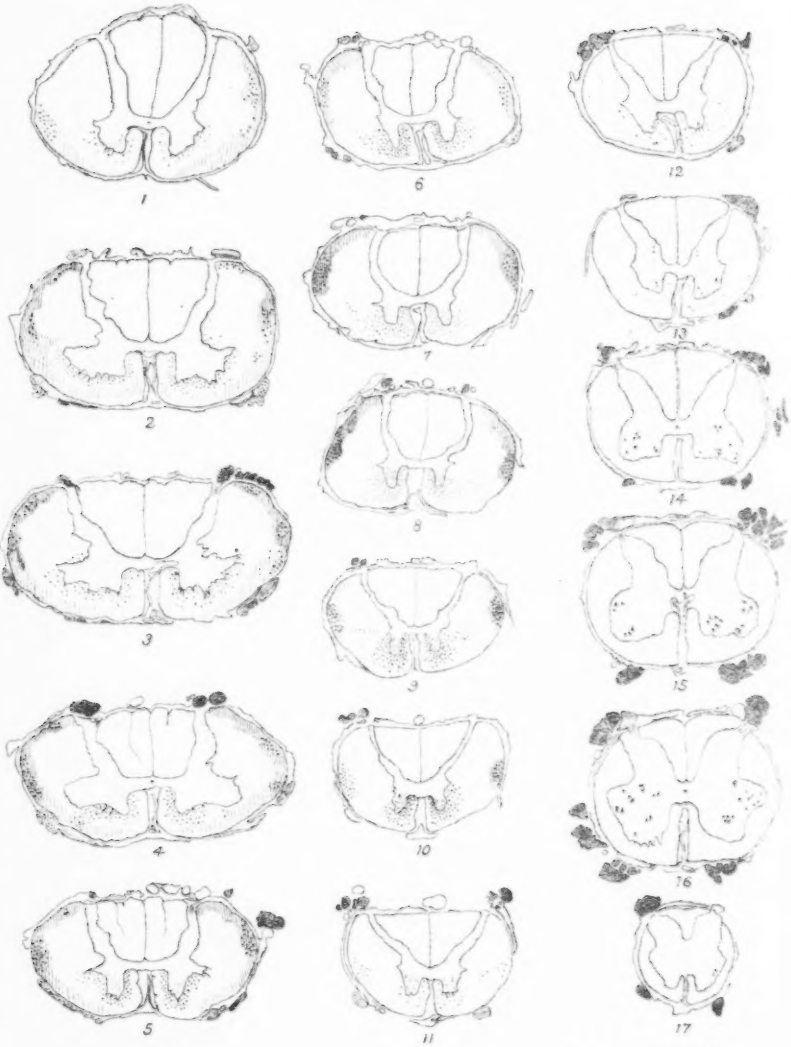
Brain.—Sections of the cortex taken from the frontal and motor region and stained by the Marchi, Weigert-Pal, and Nissl methods showed no change.

Spinal cord.—The spinal cord was examined by the Marchi, Weigert-Pal, Nissl, and van Geisson methods.

Marchi Method.

The sacral and lumbar regions of the cord showed no evidence of any recent degeneration. The anterior and

Plate I.



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posterior roots appeared normal. At the level of the twelfth thoracic segment a few degenerate fibres could be seen in the white matter around the anterior horns in the antero-lateral tracts (Pl. I., fig. 13).

At the level of the eighth thoracic segment more degenerate fibres were visible in the neighbourhood of the anterior horns, and there were also some fine black granules in the gray matter of the anterior horns. A few degenerate fibres could also be seen in the lateral region of the cord corresponding to the ventro-cerebellar tract or tract of Gowers (Pl. I., fig. 11).

At the level of the seventh thoracic segment the amount of degeneration had increased, and all round and within the anterior horns there were numerous large black granules; the number of degenerate fibres in the tract of Gowers had also increased (Pl. I., fig. 10).

At the level of the sixth thoracic segment (Pl. I., fig. 9) the degeneration had reached its maximum intensity, the gray matter being hardly distinguishable from the surrounding white matter. The degenerate fibres occupied the region of the cord all round the gray matter of the anterior horns, and to a greater or lesser extent the whole of the antero-lateral tracts, but left the crossed pyramidal tracts unaffected. The Marchi degeneration is well seen in the photograph reproduced in Plate II.—a section through the sixth thoracic segment.

At the level of the fifth thoracic segment the degeneration was still marked and occupied the same position, but was less than in sections taken from segments of a lower level (Pl. I., fig. 8).

At the level of the second and third thoracic segments the degeneration gradually became less, but occupied the same situations (Pl. I., figs. 6 and 7). There was no degeneration in the posterior columns.

At the eighth cervical segment the degeneration was still marked in the region round the gray matter of the anterior horns, but there was very little degeneration in the gray matter itself; some degenerate fibres were present in the ventral bundle of the direct cerebellar tract.

In the upper cervical segments the amount of degeneration gradually became less and less, till at the level of the second cervical (Pl. I., fig. 1) only a few scattered degenerate fibres could be seen in the white matter round the anterior horn and in the region of the direct cerebellar tract.

In the medulla the number of degenerate fibres became still fewer, those which were present being situated round the remains of the anterior horns, and some few degenerate fibres could be seen in the pyramidal tracts.

In the region of the olive there were degenerate fibres in the interolivary layer, and these degenerate fibres could be traced radiating from this point outwards. A few recently degenerate fibres were present also in the direct cerebellar tract.

In the pons no degeneration could be seen, and it may be stated that the amount of recent degeneration above the level of the second cervical segment was very little.

Nissl Method.

At and below the twelfth thoracic segment the nerve-cells of the anterior horn were numerous and stained well both with regard to the chromophyllic granules and to the nucleus and nucleolus. The cell processes were normal (Pl. III., fig. 3).

The cells of Clarke's column were few in number; they appeared swollen, and but few contained a nucleus or nucleolus. The cells varied greatly in shape and size.

In the lower thoracic segments but very few cells could be seen in the anterior horns, and such as were present were small and ill-developed.

At the level of the seventh, sixth, and fifth thoracic segments no nerve-cells could be seen either in the anterior horns or in Clarke's column.

At the level of the first thoracic a very few cells, fairly normal in appearance, were visible in the lateral horn of the gray matter.

At the level of the fifth and sixth cervical a few atrophied nerve-cells were visible; these were fewer than normal; the

PLATE II.

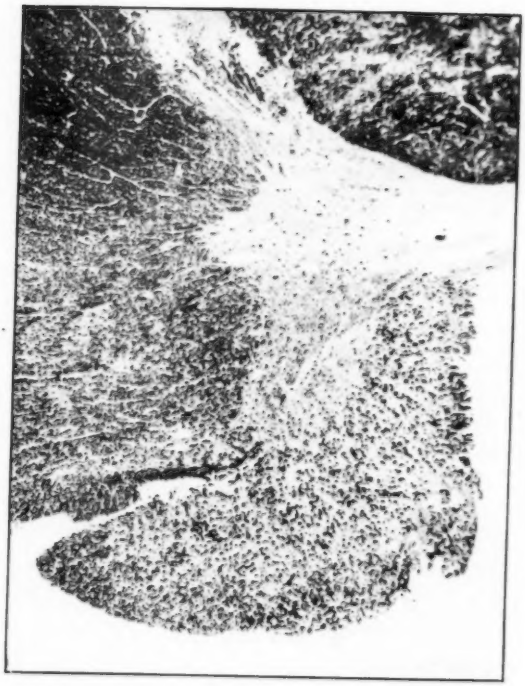

FIG. 1.

same was the case in the upper cervical segments (Pl. III., fig. 2).

In the medulla the cells of the twelfth nucleus appeared smaller and fewer in number than normal, but in other respects healthy.

Weigert-Pal Method (Pl. IV.).

Sections of the pons stained by this method showed no abnormality. In the medulla the degeneration of the direct cerebellar tract was well marked. In the upper cervical region the degeneration was most marked in the direct cerebellar tract and in Gowers' tract, while the antero-lateral tracts were somewhat less affected.

In sections through the lower cervical and upper thoracic segments the degeneration became more marked in the antero-lateral tract, and at the level of the sixth thoracic segment the whole of the ventral portion of the cord contained but few medullated fibres, and the only portions of the cord left unaffected were the pyramidal tracts and the posterior columns (Pl. IV., fig. 2).

The direct cerebellar tracts still showed considerable degeneration (Pl. IV., fig. 2).

In the lower thoracic region there was still some degeneration in the ventral portion of the cord around the periphery, but very much less than in the mid-thoracic region.

In the lumbar region some degeneration could still be seen in the ventral portion of the cord and in a small triangular area situated ventrally to the crossed pyramidal tracts. The base of this pyramid was directed towards the periphery of the cord (Pl. IV., fig. 3).

The sacral region of the cord appeared normal.

Van Geisson's Method.

Sections of the cord stained by this method showed a very marked increase of the connective tissue or neuroglia in the ventral region of the cord. (No special selective stain had been used for the neuroglia.)

The connective tissue increase was most marked at the level of the sixth and seventh thoracic segments, and diminished both in an ascending and descending direction, so that in the lumbar region there was no increase of the connective tissue.

At the level of the sixth thoracic segment no line of demarcation could be seen between the gray and white matter of the cord, the connective tissue radiating out from the central portion of the cord to the periphery, leaving only an area at the extreme periphery unaffected. This connective tissue increase affected the direct cerebellar tracts, but left the crossed pyramidal tracts and the posterior columns unaffected (Pl. III., fig. 1).

At the level of the seventh cervical the connective tissue increase was still present in the region of the antero-lateral tracts, but at the level of the fourth cervical there was but little increase of the connective tissue.

The condition of the vessels in the cord has been carefully examined. In the region of the sixth thoracic segment all the small vessels in the ventral portion of the cord were dilated and engorged (Plate III., fig. 1), and in some few places there had been exudation of blood into the perivascular space, but around none of these vessels was there any evidence of perivascular exudation of small round cells.

With regard to the blood-vessels in other portions of the cord a similar engorgement existed in the lower cervical region, but to a far less extent than in the thoracic region. The walls of the vessels, both veins, arteries and capillaries, appeared to be perfectly normal. The membranes of the cord also appeared normal.

Spinal roots.—The anterior roots of the thoracic region of the cord are very thin, and on examination but few normal fibres could be found. In the cervical region the anterior roots contained fairly numerous and well-stained fibres, while in the lumbar and sacral region the anterior roots were also well stained. The posterior roots throughout the cord appeared normal.

Nerves.—The median and ulnar nerves were examined by the Marchi, Marchi-Pal, and van Geisson methods, but

PLATE III.

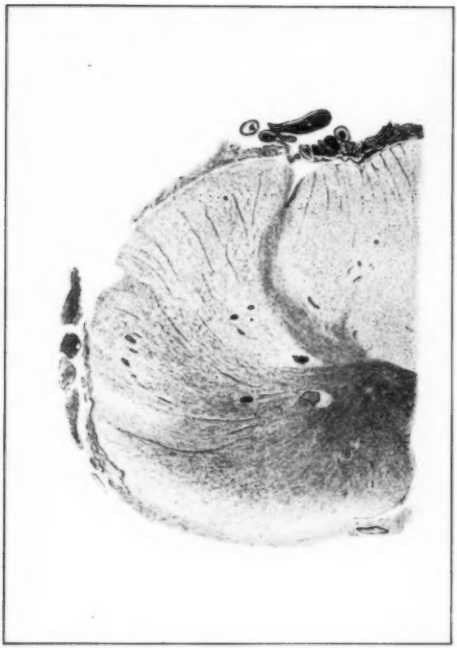

FIG. 1.

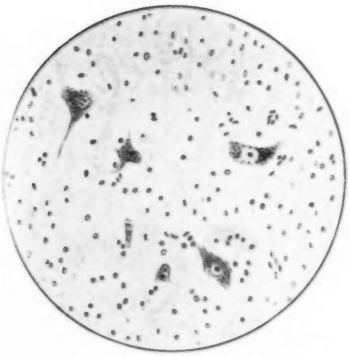

FIG. 2.

FIG. 3.

little change could be demonstrated in them either in longitudinal or transverse section.

Summary.

The changes met with may be briefly summarised as follows :—

(1) There was extensive destruction and atrophy of the cells of the anterior horn from the upper cervical to the lower thoracic segments, this change being most marked at the level of the sixth thoracic segment. The cells of Clarke's column were also affected.

(2) There was extensive degeneration of the whole of the ventral region of the cord in the thoracic segments, the lesion being most marked at the level of the sixth thoracic segment, and diminishing in extent both towards the medulla and caudally. There was extensive degeneration in the direct cerebellar tracts, both dorsal and ventral, beginning in the lower thoracic segments and extending up to their respective terminations in the cerebellum. The degeneration affected both gray and white matter alike in that region of the cord which was most affected, viz., the thoracic segments.

(3) There was a marked increase of the connective tissue in the ventral portion of the cord in the thoracic region.

(4) The walls of the vessels showed no change; there was engorgement of vessels with extravasation of blood into the perivascular spaces.

(5) The cord showed both recent and old degeneration.

Pathology.

In discussing the pathology of the above condition it is of importance to ascertain to what extent the changes present in the cord are due—(1) to a system lesion, (2) to a focal lesion, (3) or to these two lesions combined.

With regard to the system lesion the most marked degeneration of this nature is that which is present in the cerebellar tracts, both ventral and dorsal. The fact that the degeneration in these tracts begins gradually in the lower

thoracic segments and increases as the upper segments of the cord are reached points rather to a neuron degeneration than to the degeneration being due to some focal lesion in the thoracic region. In further support of this view the posterior columns and the crossed pyramidal tracts show no degeneration either in the cervical or lumbar region, and therefore the degeneration of the direct cerebellar tract is probably dependent upon some lesion of the cells of the gray matter in the thoracic segments of the cord, and not to a transverse myelitis.

If one of the thoracic segments be examined it will be found that the degeneration affects to a greater or less extent the whole of the ventral portion of the cord, and involves both gray and white matter alike. Such a condition can be explained neither on the view that there was a primary affection of the gray matter and a secondary involvement of the white matter, nor on the view that there was a primary affection of the white matter and secondary involvement of the gray matter. It seems, therefore, almost certain that the changes in the cord at this region must be due to some focal lesion.

The question then arises whether the condition could be due to some toxin acting on nerve-cells or whether the changes in the spinal cord could be produced by some vascular disturbance. With regard to a toxin acting on the nerve-cells one may at once exclude such a condition, for the destruction of the gray matter extends far beyond the nerve-cells, and that in the white matter is not limited to a neuron degeneration.

The view that the condition is of vascular origin is the only one that is supported by the pathological examination of the cord. In saying that the condition is vascular it must not be understood that the condition is of acute origin, for there is abundant evidence, both clinical and pathological, that the condition was of slow onset and progressive; there are in the same section both old and recent degeneration, both sclerosis and recent softening. No actual change can be demonstrated in the walls of the vessels. The softening has not, as in anterior poliomyelitis, limited itself to the

PLATE IV.

FIG. 1.

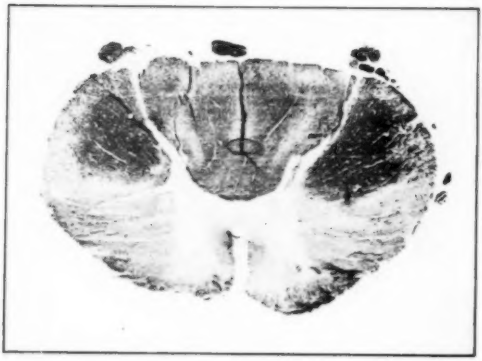

FIG. 2.

FIG. 3.

distribution of one vessel or branch of a vessel, but has involved portions of the cord receiving its blood supply from different sources, nor is there any evidence that the condition is due to thrombosis of a vessel. What is meant by the term "vascular" is that owing to some altered condition of the blood the vascular supply of some portion of the cord has been diminished or cut off, with the result that necrosis, and subsequently sclerosis, have ensued. It is suggested that some toxin is present in the blood, which not only reduces the vitality of the nervous structures, but also tends to diminish the blood supply in the peripheral portion of the circulation, and these two factors acting together produce the pathological condition found in the present case. The sclerosis which takes place results in a further diminution of blood to other portions of the cord, and the toxic condition still being present a further portion of the cord is affected, and in this way is to be explained the slow progressive process which it is suggested is of vascular origin.

One other point which is strongly in favour of a vascular lesion is the manner in which the fat present in the ventral region of the cord stains by the Marchi method, for it has been observed that the fat which results from degeneration of nervous tissue produced by a vascular lesion readily dissolves out in the xylol canada balsam in which the specimen is mounted and fades within a few weeks, whereas the fat which results from degeneration following either an experimental lesion or a neuron degeneration lasts with reasonable care with but little alteration for months and even for years. In the present case the fat dissolves out with the greatest readiness, and the specimens can only be preserved by mounting them in glycerine.

The manner in which the connective tissue spreads between the sheaths of the medullated nerve-fibres, and the fact that the gray matter and white matter are alike involved, are points which bear the most striking resemblance to the pathological process found in disseminated sclerosis; on the other hand, the distribution of the degeneration in the white matter and the character of the degeneration as shown by the Marchi method are the

points which most closely resemble the condition found in a case of combined degeneration of the cord. In combined degeneration of the cord, however, the gray matter is but little affected even in an advanced case of the disease, and the posterior columns are always affected in that disease, whereas they show no change in the present case.

We are therefore driven back to the view that the condition is a form of chronic myelitis secondary to some vascular change and toxic blood state. The origin and the cause of the peculiar distribution of the myelitis in this child remain unexplained, none of the usual factors which give rise to myelitis being present.

Description of Plates.

Plate I.

A series of sections of the spinal cord from the upper cervical to the sacral region. The sections have been drawn under an Edinger's projection apparatus from specimens stained by the Weigert-Pal and Marchi methods. The black dot represents the recent degeneration as shown by the Marchi method, and the shaded lines the old degeneration as shown by the Weigert-Pal method. The shading has not been put in in the ventral region of the thoracic segments of the cord in order not to obscure the dots representing the Marchi degeneration.

Fig. 9 is at the level of the sixth thoracic segment, and shows the greatest amount of degeneration; the recent degeneration affects the gray as well as the white matter. Below this segment the degeneration lessens, and below the twelfth thoracic segment the cells of the anterior horns resume a normal appearance.

In sections above the level of the sixth thoracic, *e.g.*, figs. 1 to 8, the degeneration in the region of the gray matter is less, whilst there is marked degeneration in the cerebellar and antero-lateral tracts.

Plate II.

Photograph of ventral portion of cord at the level of the sixth thoracic segment stained by the Marchi method. The black dots represent the recently degenerated fibres, which are seen chiefly to occupy a position immediately around the gray matter, but are also present in the gray matter of the anterior horn.

Plate III.

Fig. 1.—Transverse section of the cord at the level of the sixth thoracic segment stained by van Geisson's method. The section shows a very considerable increase of the connective tissue in the region of the anterior horns. The connective tissue radiates out from the gray matter into the white matter of the cord, and no line of demarcation can be seen between the gray and white matter in this region. There is but little increase in the number of small round cells.

No nerve element can be distinguished in the gray matter of the anterior horns, and it is only towards the periphery of the cord that medullated fibres can be distinguished. Many of the vessels are dilated, and around some of them extravasation of blood has occurred into the perivascular space. In the peripheral portion of the cord the connective tissue can be seen growing between the medullated fibres, and separating them from one another. In this respect the condition rather closely resembles the process which occurs in disseminated sclerosis, though in the way in which it destroys the nerve-cells it in no way resembles that disease.

FIG. 2.—Cells of the anterior horns from cervical region of the cord. The cells are small and atrophied, but the nucleus, nucleolus, and the chromophile granules present a normal appearance.

FIG. 3.—Cells of the anterior horns from lumbar region of the cord stained by the Nissl method. The cells may be considered normal in size and appearance.

PLATE IV.

Sections of the cervical, thoracic, and lumbar regions of the cord stained by the Weigert-Pal method.

FIG. 1.—Cervical region shows marked degeneration of cerebellar tracts, both dorsal and ventral, and considerable degeneration of the whole of the antero-lateral tract. The fibres in the region of the direct pyramidal tract are less affected, and the fibres of the crossed pyramidal tracts and posterior column appear normal.

FIG. 2.—Thoracic region. The sixth thoracic segment shows marked degeneration of the whole of the antero-lateral tracts and also of the gray matter of the anterior horns. There is degeneration also in the direct cerebellar tract.

FIG. 3.—Lumbar region. There is degeneration in a triangular area ventral to the region occupied by the crossed pyramidal tracts. It would appear from the reproduction that there is some change in the antero-lateral tracts and posterior columns, but a somewhat lighter staining has been intensified by the process of reproduction.

BERI-BERI IN MONKEYS.

BY HAMILTON WRIGHT, M.D.McGILL.

Director of the Institute for Medical Research, Federated Malay States.

In Study No. 2, vol. i., from the Institute for Medical Research, Federated Malay States, I was able to show on almost conclusive positive and negative evidence collected in the Malay Peninsula that beri-beri is independent of diet, considered as diet, and of any special food poison. That on the contrary it is an infectious disease due to an as yet unknown specific organism; that the specific organism remains virile in dark, shaded, and somewhat damp places (*e.g.*, cells of prisoners, &c.); that the activity of the organism is focal rather than widespread, because of the inimical action probably of direct sunlight; that the organism gains entrance to the body *viâ* the mouth and multiplies chiefly in the pylorus and duodenum, giving rise to a local lesion (congestion and hæmorrhagic injection of the mucous membrane, of the stomach and small gut); that it there produces a toxin which gains the circulation and causes more or less extensive bilateral symmetrical atrophy of certain afferent and efferent neurones; that the incubation period is between seven and fourteen days, and that the efficient stage of the organism in the body is between two and six weeks; that it probably escapes in the fæces of those infected, and through careless personal habit becomes deposited where it can be taken up by the fingers or where it may contaminate eating utensils, to again become ingested and give rise to the disease in other persons.

I pointed out that beri-beri from a pathological point of view could be classified under three heads, namely:—

(*a*) *Acute pernicious beri-beri*, in which the organism produced angry inflammation or marked congestion and

hæmorrhagic injection of the mucous membrane of the stomach and duodenum with other signs of infection, and a toxin of sufficient force to poison so great a number of ordinary and vital afferent and efferent neurones as to cause death suddenly or in from twenty-four hours to a week.

(*b*) *Acute and subacute beri-beri*, in which there was a lesser lesion of the stomach and duodenum, a less virile toxin elaborated, and a smaller number of ordinary and vital neurones less severely afflicted. I pointed out that cases in this category eventuated along two lines. (1) They continued in a state of partial motor, sensory, and vital paralysis for a short time after the efficient action of the organism had ceased, and then wholly recovered; or (2) they remained in a state of partial sensori-motor paralysis (the vital neurones being but slightly involved or having wholly recovered) for months or years after the active stage of the specific organism had ceased, and ultimately died of cardiac exhaustion or of the direct or indirect effects of some intercurrent disease.

(*c*) *Beri-beric residual paralysis* was the term I proposed for the second class of cases, and I endeavoured to make it clear that an erroneous estimate of beri-beri was held by many through their not having distinguished between the active stage of the disease (acute pernicious, acute and subacute beri-beri) and the stage when its effect alone was present. (Beri-beric residual paralysis).

I further showed in outline that there was a distinct difference in the underlying lesion of the central and peripheral nervous system that proved the difference between beri-beri, the specific acute infection and its continuation, residual beri-beric paralysis.

These conclusions were based on a rigid observation of the total inmates of the Kwala Lumpor gaol, where beri-beri had been epidemic or endemic for several years; on some fifty *post mortems* observed in relation to the previous history and clinical state of the cases; and on an experimental observation on healthy monkeys placed in cells that I had reason to believe were infected. I gave the full history of the observations on the prisoners and illustrative

cases of beri-beri in human beings and monkeys. The entire experimental observation on the monkeys has proved to be so conclusive that I shall set it forth here, instead of including it in a later and fuller paper on the pathology of beri-beri now in course of preparation.

To save repetition it will be well to define here the nature and stages of change observable in the various parts of the neurones in beri-beri and choose some nomenclature that will be indicative of it.

It is still an open question to my mind as to whether the beri-beric poison acts centrally on the cells, peripherally on the neuronal terminations, or on the neurones as a whole. But leaving these points aside, it can be surely stated that only the cells and the terminal portions of their dependent fibres exhibit the action of the toxin in the active stage of the disease. Later on (beri-beric residual paralysis), if the neurones do not repair, but continue to atrophy, the atrophy retrogresses and the main nerve trunks are then found to contain many markedly degenerated fibres. In no instance, however, have I found the distal ends of the posterior spinal ganglia or the ventral roots the seat of degeneration.

The earliest change I have been able to detect in the motor cells of the bulb and cord in acute pernicious or acute beri-beri is the disappearance of the Nissl-bodies of the processes and a fine pulverisation of those of the cell-body at its junction with the former. The fine homogeneous granulation may extend throughout the whole cell, or scattered discretely may be seen a few as yet unaffected Nissl-bodies. Nuclei are generally swollen and slightly eccentric. This degree of change is meant where I use the term (a) early first stage of change (see figs. 1a and 3).

It is generally the only change to be found in the cervical and bulbar cells in those cases of acute pernicious beri-beri, where after a few days of evident general paralysis or paresis of the lower limbs, the poison rapidly involves the neurones of the upper limbs and the vital neurones whose centres are in the bulb, and causes sudden death. In such cases the patient dies as the result of the poisoning of the whole mass of cardiac inhibitory and accelerator neurones. He does not

live long enough for the effect of the toxin to more than just manifest itself by a slight structural change. Many of the lumbo-sacral ventral motor cells exhibit no greater change than the "early first stage"; but the larger number are found to be in what I call here the (*b*) late first stage (see figs. 1b and 2); that is, in addition to the change already described, the nuclei stand out more clearly because of the entire disappearance of the larger Nissl-bodies, and of the fine powdery deposit derived from them. At this stage the nuclei are more eccentric than in the early first stage of change, some even about the cell border. In acute pernicious beri-beri it is chiefly the cells of the lumbo-sacral ventral horns that exhibit this stage of change. It marks a slightly longer action of the toxin on the neurones of the lower limbs.

(*c*) Second stage of change (see figs. 4, 5, 6 and 7).—By this term I propose to designate the extreme changes seen in the cells in beri-beric residual paralysis only; that is, in those cases where the paralysis caused by the action of the toxin has continued for months after the toxin itself has been eliminated. The cells in such cases are in a state of complete chromatolysis which may be dense and give the cell the appearance of being over stained, or it may be so diffuse as to render the cell nearly colourless. Along with this the nucleus is flattened against the cell membrane or bulges it out and the nucleolus seems to be about to escape. The edges of the cell may be ragged when such a stage is reached. Advanced peripheral atrophy of the Wallerian type is present in the dependent nerve-fibres.

(*d*) Third stage of change.—By this is meant the vacuolation of the cell, rupture of its membrane and of that of the nucleus, extrusion of the nucleolus, irregular staining of the whole cell, and loss or fragmentation of its processes. These are all signs of the complete degradation of the cells. It is only seen in long-standing cases of beri-beric residual paralysis or in cases of shorter standing where attempt at repair has not succeeded. It is accompanied by advanced Wallerian atrophy of great lengths of many fibres, and necessarily by the complete paralysis of movement of the parts they supplied.

Where cells are altered to any of the above degrees relational changes are to be made out by Marchi's method in the dependent fibres except where they are non-medullated. Accompanying the early and late first stage of change is a chemical alteration of the medulla of the peripheral part of nearly the whole number of fibres in the selected nerve. This is best seen in the twigs to the peronei, to the anterior tibials, the recti of the thighs and the cervical and thoracic cardiac fibres of the vagi in acute pernicious beri-beri of not more than from forty-eight to one hundred hours' duration.

The alteration is slight but general; slight because the widespread action of the toxin causes death so quickly that there is no time for a more severe Wallerian atrophy to develop.

The alteration first shows by an irregular block of staining about the nodes, with smaller irregular specks along the internodes. All internodes are affected and in inverse ratio to their distance from the organ to which the fibres are distributed. In the vagal cardiac fibres and in the phrenics, it begins to cease about half an inch from the heart and diaphragm, and ceases altogether an inch above these organs. The cells parent to fibres so affected are found to be in the early first stage of change.

In acute pernicious beri-beri of less than a week's standing it is rare for the altered myelin to break down, and form the characteristic droplets of Wallerian degeneration. When the droplets are seen they are confined to the nerve-fibres, to the peronei or anterior tibials, *i.e.*, muscles earliest paralysed. The ventral cells of those segments of the cord which give rise to such fibres are generally found to be in the late first stage of change.

In examining the neurones of those dead of acute pernicious beri-beri one is constantly struck by a desire for an antitoxin. For it is most evident that the neurones are not so much atrophied as generally slightly poisoned, and that if the poison could be quickly neutralised they would speedily function again.

Wallerian atrophy is so well known that it will be suffi-

cient if I state that where cells are found in the second and third stages of change the Wallerian atrophy of the dependent fibres is more or less advanced. Fragmentation of the axes and interstitial changes are never seen where cells are in the second stage of change (*e.g.*, in beri-beric residual paralysis of short duration), but only accompany the third stage of cellular change found in beri-beric residual paralysis of long standing.

The neuronal changes of beri-beric residual paralysis do not call for a neutralising toxin, but for the treatment meted to any advanced neuritic atrophy.

The conditions under which this observation took place have been presented together with the case of Monkey No. 27. There follows here the case histories and *post-mortem* observations of Monkeys Nos. 3, 94, 125 and 86.

Monkey No. 3.

Monkey No. 3 was a young male krah (*M. cynomolgus*) that had been at the Institute for Medical Research, Kwala Lumpor, for six weeks. During this time it was observed to be healthy. On May 13, 1902, when it weighed 8¾ lbs., it was incarcerated in cell No. 3 of the Kwala Lumpor gaol. Its diet was strictly as that of the others, *i.e.*, banana, pine-apple and sugar-cane bought in the open market. The animal remained well till July 10, 1902, when its appetite began to fail, its knee-jerks became plus, and there was hyperexcitability of the leg muscles to direct mechanical stimulation. Sensation appeared to be normal.

July 20, 1902.—There is decided weakness of lower limbs, especially of ankles. Knee-jerks can be elicited with difficulty. There is general increase of the irritability of the limb muscles on direct mechanical stimulation. Skin of lower limbs and abdomen is hyper-sensitive to pin pricks as compared with that of face and upper limbs. A slight pretibial œdema is present. The animal has a poor appetite and resents pressure of his epigastrium.

August 11, 1902.—Animal has grown slightly thinner. The muscular and sensory condition is as when last seen except that there is complete absence of the knee-jerks and

the toes; ankles and legs are about powerless. The lower arms and hands are obviously weak. There is marked wrist-drop. The animal can grasp its chain, but weakly, and when raised by it from the floor, the weight of the body is sufficient to cause the hands to relax. Several sores have appeared about buttocks within the last few days. It has proved impossible to keep them clean. The temperature of the animal has grown decidedly septic since yesterday.

August 14, 1902.—Animal weighs 8 lbs. It is nearly moribund, probably from septic poisoning. The temperature has been septic since the 10th. It is difficult to say to what extent the paresis of the lower limbs has progressed. Knee-jerks are absent. The animal does not respond to strong painful stimulation of lower limbs. Responds sluggishly to stimulation of upper limbs and thorax; quickly to stimulation of face. Heart action, rapid, weak and irregular. The animal was killed by opening his thorax. He was found to be pyæmic. There were numerous septic tubercles, ranging in size from a pin's head to a pea, in lungs, liver and spleen. The mucous membrane of the pyloric end of stomach and upper two-thirds of duodenum is slightly congested and contains a few small hæmorrhagic injections.

Microscopical Examination.

Cocci are to be seen in the spleen, liver, and kidney, and they mainly compose the small tumours in the various organs. But few cocci are observable in the spinal cord and brain. Cerebellar and cerebral cortical cells are normal; also the cells of the cranial nuclei as low down as the combined ninth, tenth and eleventh. In the lower planes of this there are from two to five cells in every section in the first stage of atrophy. The nucleus ambiguous of both sides is normal.

There are no changes in any cells of the hypoglossal nuclei or in C. segments 1 and 2. In C. 4 and 5 there are from two to five ventral cells in each section in the early first stage of change and from one to three in the late first stage. In the respiratory nucleus of Bell but few cells

are altered; never more than one or two in each section. C. 6, 7 and 8 contain a larger number in all groups in the late first stage of change. The posterior cornual cells are normal.

Thoracic 1 is not different from the lower cervical segments. In T. 2 there are proportionately fewer ventral cells affected. From here to T. 10 (T. 3, 5, 7 and 9 were examined) only an occasional cell is seen that can be said to be altered. The ventral cells of T. 10, 11 and 12 are proportionately as numerously changed as T. 1. All lumbosacral segments contain ventral cells chiefly in the late first stage of change. There are from five to nine or ten in each section. None of the posterior horn or Clarke's cells are involved.

The posterior spinal ganglionic cells of C. 1 to 5 inclusive are entirely healthy; of C. 6, 7, and 8, and T. 1 they are mostly sound. Sections in or near the median plane contain from six to twelve cells in the early and late first stage of change. T. 3, 5, 7, 10, 11 and 12 were examined. In 3 only an occasional cell shows a positive first stage of change. In 5 and 7 I detected none; in 10 about four or five in each median section, in 11 and 12 from six to ten. The lumbar ganglia and all but the last pair of the sacral were examined and found to contain from eight to fifteen cells in the early first stage, and from three to eight in the late first stage of change.

Median sections from both stellate ganglia contain from five to twelve cells in the early first stage of change. In the heart numerous Bidder's and Remark's cells are likewise affected. Sometimes all the four or five cells of a single group, or often one to three in each group.

Nerves.—The great occipitals, facials, and hypoglossals are normal. Also the twigs of the spinal accessory to the trapezius. A moderate number of the medullated cardiac fibres of the vagus are in an early stage of medullary atropy. Several small twigs close to the heart contain about one in three or four distinctly altered. In the main trunks above the clavicle no fibres are affected. The vagal atrophy does not appear to be of longer standing than between eight

and ten days. In the main trunks of the phrenics one inch above the diaphragm no changes are observable. In the smaller ramifying twigs there are about one in three or four fibres changed to the same extent as the vagal fibres. Intercostals Nos. 3, 7, and 9 are healthy, but No. 1 contains several fibres in an early stage of change. The terminal portions of the radials, ulnars, and medians all contain about one to three or four fibres in the early stage of medullary atrophy. The radials are least affected, the medians most. The twigs to the skin over the lower part of the rectus abdominis contain about one in three affected fibres. The fibres in the twigs to the skin over the rectus femoris (median cutaneous from anterior crural) are nearly all in the first stage of change. The terminal twigs of the internal saphenous to the skin over the shin, and those of the musculo-cutaneous to the skin of the dorsa of the feet, contain but few unaffected fibres. The great majority are in the first stage of atrophy. The fibres of the muscular branches of the anterior crurals to the recti and vasti are nearly all involved; some of the twigs show scarcely a sound fibre in the teased preparations. The twigs to the peronei and external longus digitorum and anterior tibials are in a similar state.

The lesion of the nervous system of this monkey is, therefore, identical with that found in man where the symptoms presenting have been within the same compass. There is no difference, so far as the microscope shows, between the character of the lesion in nerves or fibres and that seen in man where the symptoms have been of the same severity, duration, and extent.

It should be noted that the mucous membrane of the stomach and duodenum of this monkey was slightly congested and injected. The animal was killed about a month after the first observed symptom, and it is probable that an initial marked congestion was on the wane, thus marking the almost finished action of the specific organism.

I will not further discuss this point here, for I must confess that had this been the only monkey of the twelve to contract beri-beri, I should not have presented it because

of the sepsis that supervened, a sepsis which, perhaps, gave an impetus to the atrophic process already at work in the neurones. The following (one) slightly complicated case (No. 94) and (two) wholly uncomplicated clear cases of beri-beric infection will, I hope, be convincing that Case No. 3 was originally of the same nature.

Monkey No. 94.

A healthy female krah (*M. cynomolgus*), weighing 9 lbs. 3 ozs., which had been at the Institute for Medical Research for about six weeks, was placed in Cell No. 94 of the Kwala Lumpor gaol on April 28, 1902. I had reason to suspect that this cell was infected, because several prisoners who had occupied it during my 1901-2 observations had contracted beri-beri. The animal was fed entirely on banana, pine-apple, and sugar-cane. She kept well and lively until July 12, 1902, when her appetite began to fail. Only half her daily allowance of food was eaten. On examination I found her knee-jerks plus, and there appeared to be extreme tenderness of the nerve-trunks in the legs and of the skin over the tibiæ. She resented pressure of the epigastrium.

On July 21, 1902, there was decided and easily demonstrated weakness of the legs and feet. The animal could not grasp her chain as before, and took up a crouching position when at rest. Common sensation, judging from the effects of pin-pricks, was dulled over whole of the lower limbs except in a few isolated areas. The hair had almost disappeared from the legs. There seemed to be a slight wasting of the anterior tibial muscles. Trunk and upper limbs were not observably involved. The epigastrium bulged, and pressure of it was resented.

August 14, 1902.—Whole of hind limbs hairless. No sign of any kind that pinches or pin-pricks of the lower limbs are appreciated. Common sensibility of abdomen also dulled. There is marked foot-drop. The fingers appear to be weakened as they hyperextend when the animal scratches. She cannot grasp chain with feet, and when held up by

former makes only ineffectual efforts to take hold of it with hands. Sensation to pain in upper limbs seems normal, as the animal resents pin-pricks. Skin of head, neck, and thorax appears to be normally sensitive. Appetite still poor. Knee-jerks cannot be elicited; but there is hyper-excitability of limb-muscles to direct mechanical stimulation.

August 17, 1902.—Has a small abrasion on her neck from a chafing collar. It was antiseptically dressed; otherwise animal is same as in last note.

August 19, 1902.—Slight œdema of front of legs was observed this morning, and the animal had grown decidedly weaker in both upper and lower limbs. It was brought to the Institute for *post-mortem* examination before sepsis should set in. The lower limbs are almost denuded of hair; the upper limbs partially so. There was total analgesia of hands, legs, and feet; partial of thighs and arms, judging from the indifference of the animal to pin-pricks and the application of nitric acid; there were no local reflexes on stimulation. Common sensibility of abdomen was dulled. Obviously it could not be determined accurately whether or not there was æsthetic alteration, but the skin of the above parts appeared to be insensitive to the touch. Strokings of the head and pin-pricks of skin of neck and scalp called forth expressions of satisfaction or local sharp reflexes. There was decided foot-drop and partial wrist-drop. No attempt was made to grasp chain or objects placed within reach. The animal lay partly crouched on hyperextended wrists and fingers, with legs thrown out limply to right or left. Knee-jerks were totally absent, abdominal sluggish, plantar and cremasteric were not active. There was marked general increase of muscular irritability to direct mechanical stimulation. The heart beat was 142 per minute, and there were many beats so low as to be almost lost to the ear. The temperature of the animal ranged between 99 and 102 during the observation. There was no rise after the abrasion of the neck occurred. To obviate complications, however, and to determine the condition of the alimentary tract, the animal was killed by puncturing the heart at 4 p.m. on August 19, 1902.

Autopsy Notes.

August 19, 1902.—The animal weighs 8 lbs. 15 ozs. Brain and cord look normal. Right heart dilated and flabby. Stomach enormously dilated with partly digested banana. Mucous membrane of greater curvature slightly congested, also that of the pyloric end and of the duodenum and upper few inches of jejunum, and it exhibits a few small hæmorrhagic injections on the crests of the valvulæ conniventes. The duodenum and stomach are undoubtedly dilated.

Microscopical Examination.

No pigmentation or other changes in spleen or liver. No cocci in tissues. A slight fatty change can be made out in the fibres of the right ventricle by osmic acid treatment. Every third and fourth cell of Bidder's and Remark's ganglia are in the early first stage of atrophy. The vessels of congested portions of the stomach and duodenum are moderately engorged, and there are a few scattered extravasations. The cells of both Meisner's and Auerbach's plexuses of the duodenum are frequently seen in the early first stage of change. This change I never fail to find in the acute pernicious cases in man. When those attacked by the less virulent forms of the disease (acute and subacute beriberi) die while in the acute stage, the same lesion is found in them, but in lesser degree. In beri-beric residual paralysis I have never found changes in Meisner's or Auerbach's ganglia. No changes were detected in several nerve twigs to the lungs in this case.

Cerebral and cerebellar cells are all healthy-looking; so also are those of the 3rd, 4th, 5th, 6th, 7th, 8th and 12th cranial nerves. The combined nuclei of the bulb contain in their middle and lower planes about one in five or six cells in the early first stage of atrophy. These must belong to the accessory fibres of the vagus, as its cardiac branches are slightly changed. The cells of the nucleus ambiguous on both sides are healthy.

All cells of cervical segments 1 and 2 are healthy. C. 4

and 5 contain from one to two cells in each group in the early first stage of change. Several are conspicuous in the phrenic group. The ventral horn groups of C. 6, 7, 8 and 9 and T. 1 all contain cells in a more or less marked first stage of change. It is not positive that one group is more constantly affected than another. In certain sections of C. 7, for instance, the lateral or postero-lateral groups are exclusively affected, while in certain others they have escaped, and the more ventral groups are involved. T. 2, 4, 6 and 8 only rarely exhibit an altered cell. So far as I could determine during life there was no involvement of the afferent and efferent neurones from T. 2 to 8. T. 10, 11 and 12 each contain from two to four cells scattered in the different groups in principally the late first stage of change.

The lumbo-sacral segments were all carefully examined. All sections contain cells in the early and late first stage of change, but principally in the latter stage. From four to eight cells were counted of varying distribution in the different ventral groups and in the groups at the bases of the ventral horns. The cells of the dorsal horns and Clark's columns are all sound.

The root ganglia of the 5th cranial are wholly healthy, also C. 1, 2, 3, 4 and 5 ; but median sections of C. 6, 7 and 8, and T. 1, contain from ten to fifteen cells in the early first stage of change. T. 2 contains from three to seven in each median section. T. 3, 5 and 7 are to all appearances sound. T. 9, 10, 11 and 12 contain from three to six cells in the early first stage. L. 1 to 5 contain from four to eight in the early first stage, and from 1 to 3 are about as the lumbar, but 4 and 5 contain about 25 per cent. fewer altered cells. In neither cord nor ganglia is there any interstitial change or circulatory disturbance.

Nerves.—Several terminal branches of the 5th, 7th and 12th cranial and of the left great occipital were examined and found faultless. The recurrent laryngeals are also sound, and the vagi above and below the levels where they are joined by the thoracic and cardiac branches ; only a few gastric fibres are altered, but the terminations of the several cardiac twigs of both vagi that were examined show charac-

teristic early atrophy ; all the internodes and nodes of about one in two of the medullated fibres are affected, while about one in three show slighter more discretely distributed change. The terminations of both phrenics in the diaphragm contain about one in three or four fibres in the early stage of change. No change is visible in the phrenics 2 inches above the diaphragm. The terminal twigs of the median to the palmar, and of the radials and ulnars to the dorsal, skin of the hands contain an average of about one in three fibres in the early first stage of change. Twigs from the ulnars and medians to the intrinsic muscles of the hands and to the forearms all contain about one in two or three changed fibres. The 2nd, 3rd, 5th and 9th intercostals were examined and found free of change. The terminal twigs to the skin over the lower middle abdomen contain about one in four fibres all of whose internodes are affected, and about one in three where the change is more discrete.

The twigs to the lower part of the rectus femoris and internal vastus show a more advanced and more widespread atrophy than any yet described. Nearly every fibre is to some extent affected, but in no case has the atrophy got beyond the second stage. The twigs to the peronei and anterior tibials are in the same state; also the cutaneous twigs to the front of the legs from the external popliteal and internal saphenous. The cutaneous terminal twigs of the musculo-cutaneous and anterior tibials are in about the same state as the radials. There is no atrophy visible in the sciatics above the great division, and only an occasional fibre in the external and internal popliteals. Most of the branches to the posterior leg muscles contain about one in six or eight fibres in the first stage of atrophy. The cutaneous twigs from the internal and external saphenous to the skin of the calf contain but few unaffected fibres. Twigs from the external and internal plantar and from the anterior tibials to the muscles are full of affected fibres, but the atrophy is of an earlier type than that higher up, except in the case of the anterior tibials.

What should be noticed here is the large proportion of fibres affected, the confinement of the atrophy to the

terminations, and the early stage of the atrophy. It has over and over again been forced upon me that the fatality in beri-beri is due not to the extreme atrophy of a few fibres but the mild atrophy of a great many. It can be readily believed in the case of the cardiac accelerator and inhibitory fibres that if only a few were suddenly totally destroyed little harm would follow, but that when the whole number are suddenly though mildly poisoned the end is certain death.

In this case it will be observed that five weeks after the onset of the disease the congestion of the mucous membrane of the stomach and duodenum was on the wane, as in Case No. 3.

Case No. 125.

This animal, a male krah (*M. cynomolgus*), was under observation at the Institute for Medical Research for six weeks before being placed in cell No. 125 on April 24, 1902. When incarcerated it was healthy and weighed 9lbs. 3ozs. It ate well, and showed no signs of change until May 27, 1902, when I found that it had refused its food for two days. On examination its knee-jerks proved to be markedly plus and there was general marked hyperexcitability of the limb and trunk muscles on direct mechanical stimulation. There was tenderness in the epigastrium, judging by the action of the animal when that part was pressed. By June 5, 1902, the knee-jerks had vanished and there was little or no resentment shown to pin-pricks of the lower limbs. Pin-pricks of the arms and trunk were resented and gave rise to local reflex movements.

June 30, 1902.—Weakness of the lower limbs could be demonstrated, and there appeared to be total anæsthesia and analgesia of whole of lower limbs. Muscles of lower limbs very flabby.

July 7, 1902.—Weakness in hind limbs more marked. Foot-drop well developed. Appetite better. Slight wasting of anterior leg muscles. Hair beginning to fall off legs.

July 20, 1902.—As above.

July 31, 1902.—Foot-drop is marked and the legs are

almost paralysed; the thigh muscles are only slightly involved. Hands and arms are not affected. Pin-pricks of the upper limbs, trunk, and head are resented; of lower limbs are not felt. The animal regards pinches and severe pin-pricks quite complacently.

August 18, 1902.—Legs very weak. Thighs as above. Animal cannot grasp chain with feet.

August 25, 1902.—Lower limbs almost completely paralysed. There is well-marked foot-drop. No attempt is made to grip with feet. Marked increase of muscle reflex on direct mechanical stimulation. Knee-jerks cannot be elicited. The slightest pinch or pin-prick of all but lower limbs is resented, and calls forth a local reflex. Muscles of expression are normal. Heart beat 110, and markedly irregular; quickly runs to 160 after passive agitation. There are no breaks anywhere in the skin.

Autopsy Notes.

Animal killed by heart puncture on August 25, 1902. Its weight was 8lbs. 15½ozs. Slight hypostatic congestion of lower lobes of lungs was found. Stomach dilated and full of partially digested fruit. No congestion of alimentary mucous membrane. Heart appears to be normal. Spleen and liver slightly and generally congested. Kidneys normal. This animal was not killed until three months after the first observed symptom of beri-beri, and it is important to note that the mucous membrane of the alimentary tract was normal on autopsy.

Microscopical Notes.

No pigmentation of the spleen or liver, or fatty change in heart. The cerebral and cerebellar cortical cells are normal. There are one or two suspicious looking cells in the bulbar portion of the spinal accessory nucleus. Nuclei of 6th, 7th and 12th cranial nerves normal. Also nuclei of 10th and 9th. The cervical and thoracic spinal segments and ganglia as low as the 11th are sound. Sections of T. 11 and 12 show from one to three ventral cells in the

second stage, and from two to four in the early and late first stages of change. In the posterior ganglia of these segments a small percentage of cells are in the early and late first stages of change; from ten to fifteen in each median section. L. 1 to 5 and S. 1 to 3 contain a larger number of altered cells; from fifteen to twenty in the early and late first stage, and from five to eight in the second stage of change. In the ventral horns of corresponding spinal segments from one to three cells are present in the second stage, and from four to six or seven in the early and late first stages of change (see figs. 4 and 6). The stellate and a few solar ganglia were examined and found to be normal; also the right Gasserian. No intrinsic cardiac ganglionic cells were found unsound.

Nerves.—Twigs of the 5th, 7th and 12th cranial were found to be normal. In the last inch of the cervical and thoracic cardiac twigs about one in seven or eight fibres were found in the first stage of change. In the terminal twigs of the phrenics only a few changed fibres were noticeable. The main trunks of the vagi and phrenics were sound; also the terminations of the right recurrent laryngeal. No unhealthy fibres could be found in the 1st, 3rd, 5th and 9th intercostals or radials, medians or ulnars to either skin or muscle. The cutaneous terminal twigs of the anterior crurals, the internal and external saphenous, the musculo-cutaneous and the small sciatics, all contain about one in six fibres in the second stage, and one in two or three in the first stage of change. The musculo-cutaneous and middle branch of the anterior crurals are most severely affected. The ilio-inguinal was found to be healthy. The muscular branches to the rectus femoris, to the peronei, anterior tibials, and to the extensor longus digitorum of both sides are in about the same state as the cutaneous branches of the anterior crurals. Branches to the soleus contain but few altered fibres. The muscular branches of the internal and external plantars are about as those to the anterior tibials.

If the above symptoms and lesions had been observed in man, the condition would be termed rudimentary beri-beri

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(following Scheube's classification). I would classify it and the similar condition often seen in man as subacute beri-beri which, had the animal been properly treated, would have been recovered from or would have persisted as beri-beric residual paralysis. As a matter of fact the latter stage had been reached in this case, for the acute symptoms had passed off. The animal had regained its appetite, and there was no naked eye *post-mortem* appearance to suggest that there was an acute process in operation at the time of death.

Monkey No. 86.

This animal was a fine, domesticated male bruh (*M. nemestrinus*) which I got from Dr. Croucher, Colonial Surgeon, Malacca, in June, 1901. It was kept at the Institute for Medical Research, Kwala Lumpor, till March 31, 1902, when it was placed in cell No. 86 of the Kwala Lumpor gaol. Cell No. 86 I had reason to believe was infected, for during my observation recorded in Study No. 2 from the Institute for Medical Research several cases of beri-beri had originated in it.

On incarceration the bruh weighed 11 lbs., and was perfectly healthy with normal knee-jerks. It was very friendly, tame, and easily approachable. Its diet while in gaol was banana, pineapple, and sugar-cane. No rice or other dietetic articles were permitted. Water was supplied from the Kwala Lumpor supply. The animal kept well and exhibited no change whatever until August 28, 1902, when it began to refuse its fruit, became slightly irritable, and resented examination. The knee-jerks were found to be plus, and there appeared to be hyperalgesia of the lower limbs, for the animal quickly resented pin-pricks, and the latter gave rise to well-marked local reflexes. All trunk and limb muscles responded too quickly to direct mechanical stimulation. The loss of appetite continued till the animal was killed. From being a voracious eater he was content with a few bananas a day.

September 1, 1902.—Knee-jerks markedly exaggerated. Muscles of lower limbs flabby, and there is apparently slight paresis of ankle movements.

September 3, 1902.—Knee-jerks still markedly plus. Hind limbs are positively weak. Instead of sitting upright on haunches, both legs are now thrown out to left in parallel lines, and the hands, markedly hyperextended, are placed to the right to support the trunk. There is decided anæsthesia and partial analgesia of the lower limbs. The intercostals are not doing full work. When the lower limbs are passively raised they fall flaccidly.

FIG. 8.

FIG. 9.

September 4, 1902.—Early in the morning I received an urgent message from Mr. J. Galloway, the gaoler, that the animal had become completely paralysed in all four limbs during the night, and was then quite helpless. It was brought to the Institute by one of my assistants and immediately examined. Its weight was 10 lbs. 8 ozs. There was complete bilateral flaccid paralysis of the lower limbs (*see* figs. 8 and 9), marked foot-drop, and an entire incapacity

to use the legs to the slightest degree. The animal lay stretched on its right side with lower limbs fully extended and hands and arms helplessly doubled under it—the position in which it was found by the gaoler early in the morning. There was most evident flaccid paralysis of the flexors and extensors of the thighs on body, and of rotation of the hips. Wrist-drop was marked, and the complete paralysis of the fingers and forearms was beyond doubt. When the animal attempted to scratch, hyperextension of fingers and wrists was the only result. The deltoids seemed to be slightly involved, also all muscles of the shoulder girdle that could be tested. Muscles of expression seemed normal, except for their hyperexcitability on direct mechanical stimulation. He masticated slowly, but probably because of loss of appetite. Sight and hearing were seemingly normal. There was complete paralysis of common, thermal, and tactile sensation of lower limbs. Hot applications, acids, pin-pricks, were complacently regarded, and no local or other reflex followed their application. This was true also of the arms and hands, but there was only partial loss of sensations of upper arms, and of abdomen and trunk up to about the third rib. Stimulation of the face and head was appreciated by struggles to escape and scowlings. Knee-jerks were markedly plus, also the Achilles and plantar reflexes. Cremasteric and abdominal reflexes were sluggish. Respiration was altogether abdominal. The diaphragmatic jerk was lively, but the movements of the muscle were irregular, one side and then perhaps the other contracting to a maximum. There was scarcely any precardiac impulse, but the heart beat was rapid, irregular, very loud for eight to ten contractions, then sinking to almost silence. The rate at rest was 150, but after passive agitation it quickly rose to 190 to 200, and became more irregular. The pulse seemed composed of a weak, irregular wave of fine tremors. Veins in the neck throbbed. No adventitious sounds were to be heard in the lungs. The animal resented pressure of the calves and nerve trunks in the popliteal space, also pressure of the epigastrium; pressure of the hypogastrium was not resented. There were no abrasions of the skin. The

temperature ranged between 100° and 102° F. from incarceration to the time of death.

The animal was kept under observation at the Institute for Medical Research for two days. Nothing further was observed except that the knee-jerks diminished, and it became difficult to elicit them. It was found impossible to stimulate the animal to attempt to take an upright position except once or twice, when after vain trials it would fall to one or other side and lie scowling until it was petted. It ate but little, and as it began to get dirty it was killed on September 5, 1902, by opening the carotids.

Autopsy Notes.

The lungs are hypostatically congested at bases and posterior borders. There is marked acute emphysema of apices, anterior borders, and outer surfaces. Three bladders measure over 18 mm. long and 9 to 12 mm. wide. The heart is found in diastole. The right side is dilated and full of dark fluid blood. The walls are somewhat flabby. Left heart nothing of note. There are several petechiæ scattered over the surface of the right ventricle. The stomach contains a small amount of partly-digested fruit. The greater curvature and pyloric end are markedly congested, and there are numerous hæmorrhagic injections scattered over the mucous membrane and on the edges of the valvulæ conniventes. The duodenum and upper foot of the jejunum are similarly affected. Rest of gut normal. The duodenum is of large calibre and appears to be dilated. The upper mesenteric glands are slightly swollen. The spleen is slightly congested. The liver and kidneys are normal, so also are the brain and cord to the naked eye. Muscles of thighs, legs, and arms are very flabby.

The microscopical examination of this monkey was particular for obvious reasons. No cocci were found in any of the organs. The lumen of the splenic veins and capillaries are widened, and all are moderately congested. The same is true of the liver vessels. The kidneys are normal. The vessels of the mucous membrane of the pylorus and

duodenum are crowded, and there are many discretely scattered small extravasations. The muscle of the right ventricle is finely fatty, and Bidder's and Remark's ganglion cells are in the first stage of change (*see* fig. 2).

Nervous system.—The cerebral and cerebellar cortical cells are, as I have invariably found them in beri-beri, wholly normal. The nuclear cells of the 3rd, 4th, 5th, 6th, 7th and 8th cranial nerves are also normal. In the combined nuclei of the bulb there are many cells in the early first stage of change. Such cells are more numerous in the accessory and vagal portions than in the upper glosso-pharyngeal planes. In the vagal part about one in five cells are affected, in the accessory portion about one in two, and in the upper levels about one in five or seven. In the nucleus ambiguous of both sides only a few cells are affected. In some sections all are normal, in others about one in three or four are in the early first stage of change. The hypoglossal nuclei are normal.

Spinal cord.—C. 1 and 2, normal. C. 3 was partially destroyed in removing the cord, but a few of the ventral horn cells are observable in the early first stage of change. Such cells are confined to the nuclei of the accessory. Sections from C. 4 exhibit from one to three cells in the early first stage of change. Only odd sections through the phrenic nuclei show changed cells. In C. 5 a larger number of cells of the anterior and antero-lateral groups are in the early first stage of change. The larger number of the sections show changes in the cells of the phrenic nuclei. In C. 6, 7 and 8 and T. 1 the majority of the ventral horn cells are affected; from three to eight in the early first stage and from one to three in the late first stage. No posterior horn cells of the segments just reviewed are in any way altered. The lateral ventral horn cells of T. 2, 4, 6, 8, 9, 10, 11 and 12 are nearly all in the early first stage of change. A few of the more anterior cells are in the same state in T. 2. In T. 9, 10, 11 and 12 a larger number are in either the early or late first stage of change; from one to three in the latter and from one to seven or eight in the former stage. Posterior horn and

Clarke's cells are normal. All the ventral groups of the lumbo-sacral segments contain cells in the early and late first stage of change (*see* fig. 3), from three to ten in the former and from one to three in the latter stage. Posterior cornual cells of this region are all normal. All posterior spinal ganglia below C. 2 contain numerous cells in the early first stage of change, and a smaller number in the late first stage. C. 6, 7, 8 and 9, T. 1, 11, 12, L. 1 to 5 and S. 1 to 5 are most obviously involved. Sections through the median planes of the ganglia contain from ten to twenty-five altered cells (*see* fig. 1). It is of great interest that the stellate ganglia are rich in cells in the early first stage of change. In median planes of the ganglia every second cell is involved. I invariably find this to be the case in acute pernicious beri-beri in man. It is a clear indication that the non-medullated accelerator fibres to the heart are poisoned equally with the vagal inhibitory. Such a state of affairs is alone sufficient to account for death in cases of acute pernicious beri-beri. Several of the solar ganglia were examined, but only a few cells seemed abnormal. A few fibres of the plexus are in the early first stage of change. I have never found the cells or fibres of the solar plexus affected to any great extent, except where marked œdema was a symptom.

Nerves.—Those parts of the 5th, 7th and 12th cranial nerves examined are normal in their terminations and in the middle of their course. There are a few fibres slightly changed in the pharyngeal plexus to the upper part of the pharynx, but whether fibres of the 9th or 11th I cannot say. The terminations of the left recurrent (vagal motor fibres from the spinal accessory) contain about one in three very slightly altered fibres. The cervical and thoracic vagal fibres to the heart are nearly all slightly affected for the lower inch or half inch of their course. The second stage of atrophy is not apparent in any of them. The vagi are quite free of atrophy below and above the points of departure of the cervical and thoracic cardiac fibres and between the latter points. This is a remarkable example of the truly selective and peripheral effect of the beri-beric

toxin. The terminations of the great occipital to the scalp are unaffected. The terminal twigs of the radials, medians and ulnars to muscles and skin are almost without exception more or less slightly changed. No single fibre is greatly changed, but none have escaped. This state of affairs is to be observed in the intercostals examined; namely, T. 1, 3, 5, 9 and 10. The terminal twigs of the phrenics have all been lightly touched by the toxin, but less so than the intercostals. The terminal twigs to the skin over the lower abdomen are also slightly but generally affected. The fibres of the lateral, median and external cutaneous branches of the anterior crurals are all involved, most of them to the late first stage of change. The anterior branches of the external popliteal and internal saphenous to the skin of the legs and the cutaneous branches of the musculo-cutaneous are likewise affected. The twigs of the external popliteal and internal saphenous to the skin of the back of the legs are less obviously affected; the change is entirely early first stage. The twigs of the internal and external plantars to the skin and intrinsic muscles of the foot are all affected, chiefly to the late first stage. The twigs to the posterior leg muscles are almost all in the early first stage. But those to the vastus internus, rectus femoris and peronei, are nearly all in the late first stage. A few of them exhibit numerous globular masses of altered myelin.

Conclusions.

If there is any doubt about the preceding cases being beri-beri, Case No. 86 ought to most surely dissipate it. The lesion of the nervous system is characteristic of acute pernicious beri-beri as seen in man.

The onset, early and developed symptoms, the underlying lesion in the stomach, duodenum, and nervous system, the conditions under which the disease was contracted, are so similar to what I have found in regard to man that I have not the slightest hesitation in concluding that all these monkeys contracted beri-beri. Case No. 3 alone would not have warranted such a conclusion, not because the animal

had become septic, but because it had become so grossly septic.

It could not be urged that beri-beri in man was not beri-beri because a few days before sepsis had occurred as the result of infection through bed or other sores, or because of death being due to some intercurrent disease such as phthisis or dysentery. No doubt sepsis and intercurrent diseases may modify to some unrecognisable extent the characteristic lesion of beri-beri in the peripheral and central nervous system, but that is all. On the other hand there is no doubt whatever that the lesion of the nervous system in beri-beri induces disease in the lungs and other parts of the body, *e.g.*, phthisis, fatty degeneration of the heart, liver, &c.

The fact, therefore, that Case No. 27 already published, and Case No. 3 included here, were septic during the few days before they were killed does not militate against my conclusions, for the characteristic symptoms of beri-beri preceded the sepsis by weeks.

I would classify Cases No. 27, 3 and 94 as acute, and 125 as subacute beri-beri that would have recovered or have ended as residual beri-beric paralysis had the animals not been killed during or shortly after the active stage of infection had ceased and the organism and its toxin eliminated.

But Case No. 86 I would classify as acute pernicious beri-beri in which the causal organism developed rapidly, producing so virulent a toxin that it acted widely and severely on the vital and ordinary afferent and efferent neurones, and so rendered the animal almost moribund in a few hours. Had it been given a chance I do not think this animal would have recovered.

The value of this observation lies in the fact that it conclusively proves:—

(*a*) That beri-beri is an acute infectious disease.

(*b*) That the organism—as yet not known—exists in close sunless foci, *e.g.*, the cells of a gaol in this instance.

(*c*) That no food as food, either qualitatively or quantitatively, is a factor in the production of the disease.

(*d*) That the organism is not an organism that affects or developes on food commonly used by man.

(*e*) That the organism is probably ingested with food accidentally contaminated by it.

(*f*) That it multiplies in the stomach and upper part of the small gut, causing a local congestion or inflammation, and elaborates a toxin which, being absorbed, acts with varying force bilaterally and symmetrically on certain vital and ordinary neurones to give rise to the collection of symptoms known as beri-beri.

(*g*) That the incubation period of beri-beri is short.

(*h*) That rice, either as diet or as the habitat of a living specific organism, cannot for a moment be seriously considered.

These conclusions were arrived at after a careful experimental study of the disease in man, and is confirmed by this observation on monkeys. It but remains to detect the organism.

I am convinced that no good but only confusion will result if bacteriologists and other students of the subject do not firmly grasp the distinction I have made between beri-beri, the acute infectious disease, and its residual paralysis.

Explanation of Plate.

Fig. 1.—Posterior spinal ganglionic cells from L. 4, monkey No. 86; (*a*) early first stage of change; (*b*) late first stage of change.

Fig. 2.—Group of cardiac ganglionic cells from right heart of monkey No. 86, in early and late first stages of changes.

Fig. 3.—Cell from the antero-lateral group of the ventral horn of L. 4, of monkey No. 86, in early first stage of change.

Fig. 4.—Cell from ventral horn of ventral group of S. 1, monkey No. 125, in the second stage of change.

Fig. 5.—Cell from phrenic nucleus of C. 5, monkey No. 27, in second stage of change.

Fig. 6.—Cell from ventral horn of L. 2, monkey No. 125, in second stage of change.

Fig. 7.—Cell showing second stage of change from ventral horn of L. 2, monkey No. 27.

Fig. 8.—Monkey No. 86. The almost complete flaccid paralysis; foot and wrist-drop are apparent.

Fig. 9.—Monkey No. 86. The complete helplessness of the animal is well shown.

ASSOCIATED MOVEMENTS IN HEMIPLEGIA: THEIR ORIGIN AND PHYSIOLOGICAL SIGNIFICANCE.

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An involuntary association between the movements of paralysed limbs and other parts of the body is a well known thing in cases of hemiplegia. When, for instance, the leg on the unaffected side is voluntarily moved, it often happens that the paralysed leg moves with it, and when a deep yawn occurs the paralysed arm is frequently drawn across the chest with great force. While the mechanism by which these movements are probably produced has been carefully studied the physiological reasons for their existence do not appear to have received so much attention, and, after briefly referring to their mechanism, I shall endeavour to show that, at any rate in some cases, these movements have a definite history in evolution, and that they were once useful for the ordinary purposes of life.

In the case of a complete hemiplegia due, say, to destruction of the internal capsule, it is instructive first of all to enquire what changes have resulted from the lesion in order to allow the associated movements to take place, or, to put the question in another way, to enquire what influences are present in health to prevent them taking place.

The answer to this question is to be found by a consideration of the functions of the pyramidal tracts which are destroyed in such a lesion as that suggested above.

These functions are in the main twofold; the fibres

conduct (1) impulses which give rise to voluntary movements, and (2) impulses which exercise a constant inhibitory effect on the lower centres. When these fibres are destroyed there is loss of voluntary power as seen by the paralytic symptoms, and loss of inhibition as seen by the rigidity and increased reflex irritability. It is doubtless this loss of inhibition which allows associated movements, for otherwise there seems no reason why those which occur in hemiplegia should not also occur in health.

With regard to inhibition in the case of the respiratory centre, and the results of cutting it off in hemiplegia, Dr. Hughlings Jackson's observations may be referred to here.[1] In hemiplegia Dr. Jackson found that during ordinary or automatic respiration the amplitude of the superior intercostal movements was greater on the paralysed than on the sound side, while in voluntary respiratory movements the movements were greater on the sound side. Pierce Clark[2] has recently confirmed this observation and gives the following explanation. "In destructive lesions of the internal capsule causing paralysis, hemiplegic in character, the cortical inhibitory control over the medullary respiratory centre of the paralysed side is destroyed in greater part, at least much more so on the hemiplegic side than to that centre presiding over the sound side; in consequence of this withdrawal the uninhibited medullary centre overacts in automatic or ordinary respiration, hence excess movements on the paralysed side. But in forced conscious or volitional (cerebral) respiration the movements of the thorax on the opposite sound (or nearly so) side are greater."

It may then I think be safely inferred that the first step in the generation of pathological associated movements is the loss of inhibitory impulses.

Normal inhibition being removed, the second step consists in the simultaneous action of the cells on the paralysed

[1] *Lancet*, February 23, 1895.
[2] *Am. Jour. Med. Science*, December, 1903. See also some remarks by Dr. Judson S. Bury, *Lancet*, December 19, 1903.

side with those with which the movement is to be associated, thus the cells will act with their fellows of the opposite side in the associated movements of the two legs, or with the cells connected with the respiration in the case of the movement of the arm during yawning. On this part of the subject Sir William Broadbent's classical observations have thrown some considerable light.[1] These observations were made with a view to explain the escape of certain movements in hemiplegia, and Sir William Broadbent found that the key of the solution lay in the fact that the movements which escaped were those which habitually acted with their fellows of the opposite side, and he attributed this immunity to the existence of free commissural paths between their nuclei, so that impulses could pass from the nucleus on the sound side to the nucleus on the paralysed side. In other words, an impulse from either side of the brain would, through the commissure, reach both nuclei and so cause the bilateral movements to be saved, when the unilateral movements, which have no such convenient commissures to help them, have perished. This hypothesis, which has proved of such importance in the study of neurology, will practically explain the actual mechanism of the post-hemiplegic movements. The two stages in the development of these movements would clearly be (1) the removal of inhibitory influences on one side by destruction of the pyramidal fibres, and (2) action in conjunction with adjacent nuclei, brought about by impulses passing along commissural fibres. I would again lay stress on the fact that the first step must be the removal of inhibition, which has gradually become developed as the education of the limbs for isolated and special movements has proceeded, for had this inhibition not been developed both legs would probably move simultaneously like the movements for closing the eyes. Another point to be observed is that the constant presence of inhibitory influences leads to gradual disuse of the commissural fibres until finally it becomes difficult, or even impossible, for impulses to pass along them

[1] *The British and Foreign Medico-Chirurgical Review*, 1866.

at all. This effect is well seen if we again for a moment compare the effects of a hemiplegia on movements for closing the eyes with movements for the legs. Those for closing the eyes are scarcely materially altered, for the two sides have not yet become educated to act independently of one another; the commissural path between their nuclei is easily traversed, and the nuclei are in a condition to receive and to act from the impulses that come along it. On the other hand, the legs are so much in the habit of being moved separately that their commissural fibres have become disused, and when the inhibition is taken off, as it is in hemiplegia, the disused paths are of but little use and the movements of the limb suffer in proportion.

But while Sir William Broadbent's theory accounts for the mechanism of the movements after inhibition has been removed, it will be noticed that so far from all these movements being beneficial like those of closing the eyes, they are often useless and even actually detrimental. Only to be able to move the two legs together, though possibly a little better than not being able to move them at all, can be of very slight use, and it is certainly both useless and unpleasant to have the arm drawn across the chest during the act of yawning.

The commissural fibres which allow these movements to take place must once have had their uses or they would not have been developed, and therefore it is reasonable to suppose that impulses which were of some importance once flowed along them, and that as these impulses ceased the paths gradually became disused. When, however, the influences that keep the impulses from flowing along them are removed, the old disused routes are again traversed although the results may no longer be advantageous to the individual. These associated movements of hemiplegia may then be looked upon as a faint reflection of movements which have gradually become obliterated in the process of evolution, and I shall now endeavour to trace out how these movements originated and what purposes they previously served. In the first place evidence seems to show that all movements were originally bilateral. The

passage of bilateral movements into unilateral ones can be easily traced in man, and in every one, many movements which were primarily associated tend to become dissociated, and the dissociated ones then become still further specialised for particular purposes.

We have only to compare the arms and hands of man with the forelegs and feet of animals to realise this.

Thus the question of loss of power in hemiplegia may be approached from another standpoint, and we may ask, not why some movements escape in a unilateral lesion, but why some are lost, for working upwards from the time when all movements were bilateral it will be seen that the escape of movements in a one-sided lesion would be the natural thing to happen, and that their loss is the result of a gradual dissociation and specialisation. The involuntary associated movements of the two legs in hemiplegia are, according to this supposition, the representation of the bilateral movements of the legs which at one time served as the chief means of progression.

When bilateral symmetry was first attained (which it was at apparently a very early period of animal evolution), all movements including those of the limbs were doubtless bilateral, and this mode of progression still remains in various animals, as in the leaping of frogs and the hopping of many birds. Progression by alternate movements of the legs is a later attainment, and the tendency to revert to the more primitive method may often be seen in animals when they are under the influence of excitement. The horse and the bull, for example, usually walk or trot, but when angry or frightened they tend to gallop. As the art of walking by separate movements of the legs is acquired, the movements of the two forelegs become associated with those of the two hind legs, but in such a manner that the left fore-limb moves in conjunction with the right hind limb and the right fore limb with the left hind limb. This diagonal association of the limbs is still seen in man at the present day by his tendency to swing the arms alternately with the legs in walking. To abstain from swinging the arms in this way is an effort and can only be accomplished

with some trouble.[1] Man has entirely lost the more primitive mode of progression by the simultaneous movements of both legs, and he never apparently finds any reason to resort to it unless it be when he jumps, and even here in most cases one leg tends to hang behind the other. Such a mode of progression would be of no advantage to him after he assumed the upright position. But although obsolete in health the old association shows itself again when the inhibitory influences which check it are removed by disease.

When the inhibitory impulses cease to exist the memories of the old bilateral movements assert themselves, but the paths have become difficult to traverse and only a feeble impulse can get through to produce a correspondingly feeble movement.[2]

The circumstances which give rise to involuntary movements of the arm during yawning are not quite so obvious, for it is not apparent at first sight why the two movements should have ever been associated.

That the occurrence of these movements is dependent upon the removal of inhibitory influences is clear from what has already been said, and it is interesting to note that the dissociation has not yet become quite complete even in health, for the involuntary stretching of the limbs during a yawn is familiar to every one.

In one instance I have observed that deep yawning is occasionally accompanied by some involuntary jerky movements of flexion of the left arm, quite different to the ordinary stretching of the limb and much more nearly approaching the movements one sees in hemiplegia. It is also interesting to note that in this case it was the left

[1] Sir William Broadbent alludes to this point in his recent "Hughlings Jackson" Lecture (BRAIN, Pt. ciii.). He says: "I think a further exemplification of the principle (*i.e.*, the bilateral association of nerve centres) exists in the cord, in fibres which may be assumed to extend from the arm centre of one side to the leg centre of the other or *vice versâ*, which have to do with the automatic swinging of the arms in walking and running; which may, moreover, have to do with the imperfect paralysis of the leg in hemiplegia, and which certainly, in my opinion, give the explanation of the circus movement of the dog instead of hemiplegia in severe lesion of the motor area."

[2] It seems probable that the comparative escape of the leg in hemiplegia is due to the existence of these commissural fibres rather than to those connecting the arm and leg as suggested by Sir William Broadbent (*v.* preceding footnote).

arm which moved, for in a right-handed man one would expect the left arm to be less specialised and therefore less strongly inhibited than the right.

In hemiplegia the paralysed arm is often drawn across the chest with very great force and at the same time the leg may be drawn up. In order to arrive at some conclusion with regard to the meaning of this association it is necessary to consider some aspects regarding the development of the function of respiration.

If we go back to the fish we find the lungs represented by a swim-bladder, the chief use of which is to allow the fish to vary the depth of its position in the water. Compression of this bladder appears to be accomplished usually by the lateral muscles of the body, though in some cases there is a distinct muscular coat developed in the bladder wall. The two pectoral and the two ventral fins are the homologues of the future limbs, and it is interesting to note that the chief function of these is not yet one of progression, but rather of balancing the body of the fish, and in performing this duty these fins appear to act to a considerable degree in connection with the condition of the air bladder. Cuvier says: "The pectoral and ventral fins do not appear of much use in the progress of fishes, but they employ them to preserve themselves in a state of equilibrium or a state of rest, and they extend them whenever they find it necessary to correct the vacillations of the body."

The nerve supply of the air bladder in fishes is the vagus, as it is that of the lungs in later development, and in some species this nerve sends fibres to the pectoral and ventral fins from its lateral branches. There are then even in fishes, before any lung respiration is developed, some signs of association between the representatives of the future limbs and the future lungs.

Lung breathing appears at first to be developed as a supplementary aid to branchial respiration, and the fish or amphibian that cannot obtain sufficient air from his watery surroundings seeks more by coming to the surface of the water and swallowing it.[1]

[1] (*v.* "Principles of Biology," by Herbert Spencer, vol. ii., p. 835).

Repeated deglutition of air bubbles finally results in the differentiation of the lungs from the swim-bladder, but it seems obvious that as soon as the animal begins to feel the benefit of exchanging its air in this way, and habitually practises this method of obtaining it, the getting rid of the air must be quite as much a voluntary effort as taking it in. It is only later that the respiratory acts will become controlled by automatic centres. Respiration, more especially expiration, will no doubt at first be effected by the trunk muscles, and the nervous mechanism employed will not at that time be specially differentiated, and it is probably these early centres further developed which survive to the present day as subsidiary respiratory centres in the cord, of which more will be said later.

In making these early expiratory efforts the animal would find it necessary to have some fixed point for the muscles to act from and this would naturally be obtained by fixing the limbs, a piece of knowledge which is inherited by man and put to practical use when he voluntarily brings his extra respiratory muscles into play, as in asthma.

Another interesting piece of evidence concerning the association between movements of the limbs and respiration is furnished by the Chelonia, to which class belong the turtles and tortoises. In Owen's work on comparative anatomy it is stated that the lungs of these animals are lodged in a cavity the capacity of which is only affected by the retraction and protrusion of the limbs and tail, and that if a turtle is thrown upon its back, and makes an inspiration, its four feet are erected, the breast bone is pushed forward, and the cavity swells out wherever the parts are soft, and it is suggested that all this is done by the muscles of the extremities moving their respective bones in an inverted order; that is, the extremity becomes the fixed point from which the other bones move.[1]

Next, it will be well to consider the subsidiary respiratory centres that still exist in the cords of mammals, and which, no doubt, represent centres that at one time took a very

[1] Respiration can also be carried on when the limbs are motionless, apparently by acts of deglutition. *v.* Owen's "Comp. Anat."

important, if not the chief, part in the functions of respiration. Sir Michael Foster, in his text-book of physiology, states that in young animals, under certain conditions, respiratory movements may be carried out with entire absence of the spinal bulb, and that these rhythmical respiratory movements are sometimes accompanied by rhythmic movements of the fore and hind limbs.

It should, however, be stated that Sir Michael Foster for some reason does not consider that these movements of the limbs are respiratory in action, and owing to their presence he doubts whether the experiments really prove the existence of distinct respiratory centres in the cord, but unless there is any special reason for thinking otherwise it would seem reasonable to believe that the movements in question represent an association between the movements of the limbs and those of respiration. Moreover, to some authorities these centres have a higher significance, for Sir Michael Foster goes on to say that some consider them to be the chief centres which the bulb merely governs. Anyhow, this was probably their position at one time, until they gradually lost their independence as they came to be more and more under the control of the higher centre.

Because these centres are unable to carry on respiration when the bulb is destroyed it must not be too hastily assumed that they are necessarily so useless when the mechanism is complete, for it may be that under the influence of the bulb they discharge duties which they are unable to do as soon as that influence is removed.

To sum up, then, it would appear that the evidence goes to show that respiratory movements in animals were originally closely associated with movements of the limbs, and it seems likely that it is this association that accounts for the presence of commissural fibres between the nuclei which preside over the two movements. As the respiration became more thoracic in character its association with the legs would become weakened, and then, later on, the movements of the arms would gradually become dissociated as they became educated to perform more special movements, for the inhibition required for these finer movements would

effectually prevent any movement taking place with respiration, which, if allowed, would obviously destroy the utility of the arms for other purposes. At the same time the increasing automatic action of the respiratory movements would also tend to make ordinary respiration independent of any support from the limbs except in cases of difficulty, and thus the movements of respiration and of the limbs, at first intimately associated, would gradually drift apart until finally, with the development of inhibition on the one hand, and with the disuse of the commissural paths on the other, the association would practically altogether disappear, but as soon as the inhibitory impulses are taken off the limb again tends to resume its old relations, the unused paths become reopened and the old associations are revived. In health these paths have still, in this instance, been used a little during yawning, so that their obliteration has never been quite complete. Yawning is a peculiar respiratory act in many ways and is certainly more automatic than ordinary breathing. According to Sir Michael Foster the stimulus in yawning probably acts upon the respiratory centre itself; possibly the centres in the cord also take a share in it, but in any case yawning may be looked upon as a lower type of the respiratory process than ordinary respiration, and therefore the one with which the movement of the arm would be likely to be longest associated.

Darwin's first principle in his work on "The Expression of the Emotions" is "that movements which are serviceable in gratifying some desire, or in relieving some sensation, if often repeated, become so habitual that they are performed, whether or not of any service, whenever the same desire is felt, even in a weak degree." I would then suggest that these post-hemiplegic associated movements are in keeping with this principle inasmuch as they represent movements which were once serviceable, but which have now ceased to be so. These movements, though scarcely discernible in health, have not, however, yet become entirely obliterated, and they are still able to show themselves clearly when the restraining influences that normally check them are removed.

EXPERIMENTAL RESEARCH ON THE COURSE OF THE OPTIC FIBRES.

BY GEORGE DEAN, M.B., AND C. H. USHER, M.B.

(*Second Communication.*)

In a previous communication to the Ophthalmological Society[1] we reported results obtained by us in rabbits, which showed that a degeneration of the optic nerve resulted from a lesion of the retina; that the degeneration had a limited distribution, occupying in the main the same position in relation to the nerve section throughout the whole length of the nerve, and that the position of the degeneration in the sectional area of the nerve corresponded with the part of the retina wounded. The degeneration in the optic nerve resulting from a macular lesion in the monkey was also described.

The lesions of the retina were made by a Graefe knife. Marchi's osmium-bichromate method was used for demonstrating the degeneration. For the details of the methods of operation, fixation, hardening, and staining, we must refer to the previous communication.

In the present communication we propose to give the results of other experiments on monkeys in which degeneration (*a*) in the optic nerve, and (*b*) in the chiasma, resulted from retinal lesions or division of an optic nerve. The method of operation on the retina was the same as that adopted in the case of the rabbits, namely, entrance of a Graefe knife at the posterior part of the ciliary region, guidance of the point of the knife, aided by the ophthalmoscope, and incision of the selected part of the retina, or the

[1] Vol. xvi. of the *Transactions*. Please note that figs. 52 and 62 are drawn as seen by the indirect method, and "D" and "V" should be transposed in fig. 67.

production of a lesion by means of a fine thermo-cautery, as in the experiment on the monkey described in our last communication.

The methods used for hardening and staining the tissues, and also those for keeping the optic nerves from rotating, were the same as those described in the previous paper.

A complete series of transverse vertical (coronal) sections was made of the chiasma in each case.

The cases dealt with in the present communication can be conveniently divided into three groups:—

(I.) Division of an optic nerve.

(II.) Retinal lesions at a distance from the macula.

(III.) Retinal lesions at the macular region.

Group I.—Division of an Optic Nerve.

In each case the nerve was divided in the orbit. Attempts were made to accomplish this in such a way as to do a minimum of damage to the blood-vessels.

Monkey 1 (Macacus Rhesus).

In this case the left optic nerve was divided transversely far back in the orbit. The animal lived only eight days, and the cause of death was uncertain.

No ophthalmoscopic examination was made after the operation.

Microscopical examination of the optic nerves.—In front of the incision there is well-marked degeneration as evidenced by numerous black points throughout the section. Behind the incision there is little or no evidence of degeneration; no black points can be made out.

Monkey 2 (Macacus Rhesus).

The right optic nerve was divided far back in the orbit. The animal was killed after thirty-two days.

Ophthalmoscopic examination before killing the animal: optic disc pale, retinal vessels not easily made out.

Microscopical examination.—In front of the incision there is a considerable amount of new fibrous tissue and comparatively little staining by osmic acid. There is an

entire disappearance of the normal relation of the framework of the nerve and its nerve bundles. Behind the incision there is dense staining by osmic acid throughout the sections.

Monkey 3 (Macacus Rhesus).

The right optic nerve was divided far back in the orbit. Immediately after operation the retinal arteries were reduced to the size of fine threads. Retina somewhat hazy. Retinal veins not markedly changed. The animal lived fifteen days.

Microscopical examination.—In front of the incision the vessels of the septa are very prominent, and the septa distinct. There are numerous black points throughout the whole area of the nerve. It was observed that the transverse diameter of the nerve was greater in front than behind the incision.

Behind the incision there is a diffuse well-marked degeneration containing more densely-packed black points than in front of the incision.

Chiasma.—On tracing the degeneration from before backwards it is found in front to be almost confined to the right half; on the ventral aspect, however, there is a narrow tract passing to the left side.

Further back the crossing degeneration is much more extensive, and is arranged in distinct transverse columns. Still further back other degenerated columns appear and occupy a region nearer the dorsal surface, and there is a widespread degeneration on both sides of the chiasma, but the degeneration remains much less marked in the left lateral region.

On the right side of the chiasma posteriorly, and occupying a ventral position, is an area presenting very few black points; the corresponding area on the left side is densely packed with them.

Monkey 4 (Macacus Rhesus).

The left optic nerve was divided far back in the orbit. Immediately after operation the optic disc was pale, and the retinal arteries much narrower than those of the other eye.

The animal lived thirty-one days.

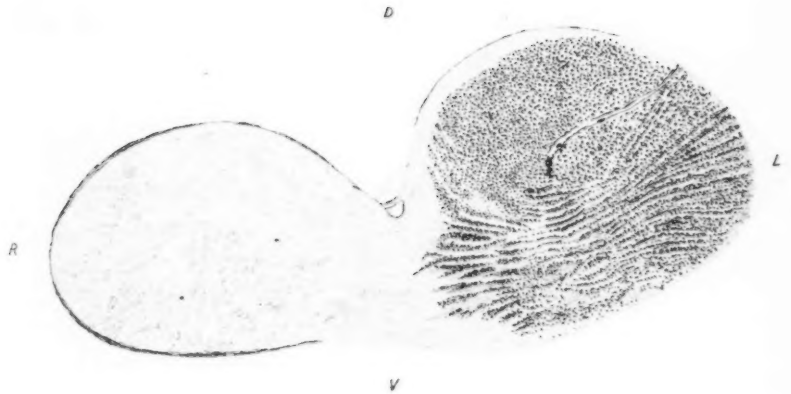


Fig. 1.

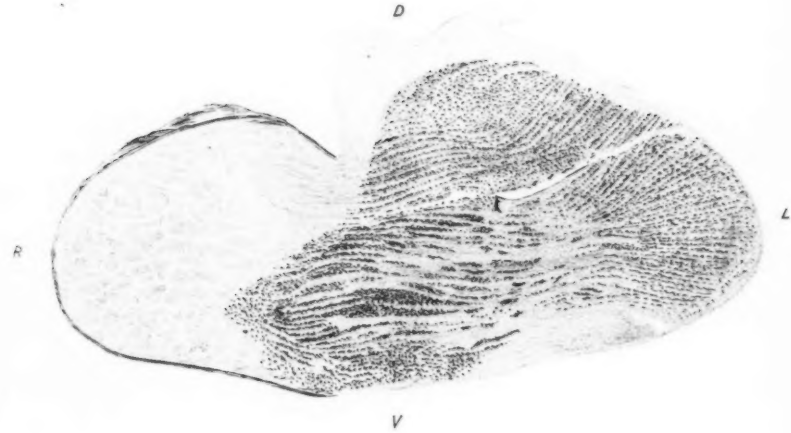


Fig. 2.

Bale & Danielsson Ltd, Lith.

Microscopical examination.—Behind the scar there is a dense diffuse degeneration. In front of the scar there is a degeneration, but the black points are smaller than behind the scar. The normal relation of the fibrous tissue to the bundles is destroyed, the angular character in these having disappeared, the bundles being now rounded in outline.

Chiasma (Plates I. and II.).—In the front part of the chiasma the degeneration begins to cross at once on the ventral aspect. Further back it encroaches more and more on the right side, most of it still lying in the ventral part. The black points are here arranged in transverse bands. The crossing degenerated fibres become more numerous and fill more and more of the dorsal part.

Towards the posterior part of the chiasma a ventral median area on the same side as the operated nerve becomes free from degeneration, and further back this area increases in extent. The corresponding area on the other side is densely packed with black points. In sections from further forward in the chiasma this region (ventral median) contains fewer degenerated fibres. The outer part of the chiasma on the side of the unoperated nerve is practically free from black points. The outer part on the other side is full of black points.

Monkey 5 (Macacus Rhesus).

This was an enucleation of the right eyeball. The animal lived thirty days.

Microscopical examination.—There is well-marked degeneration throughout the nerve.

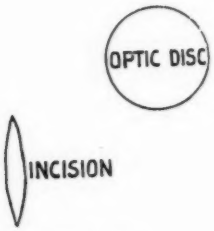
Chiasma.—The crossing of the degeneration in the chiasma commences on the ventral part anteriorly, and on passing back gradually involves an increasing area of the left side. At the posterior part of the chiasma on the right side the ventral median area contains very few black points. The corresponding area on the opposite half of the chiasma is very full of black points. The degeneration is much less marked at the outer part of the left half. Posteriorly no marked difference as to the number of degenerated fibres can be made out between the two sides.

GROUP II.—RETINAL LESIONS AWAY FROM THE MACULA.

Monkey 6 (Macacus Rhesus).

An incision was made in the right eye below the macular region with a Graefe knife.

Ophthalmoscopic appearance immediately after withdrawal of the knife.—There was a bright red area, one and a quarter discs' diameter in length, and its breadth one-sixth of its length. It had well-defined margins, its long axis was vertical, and was so situated that its upper end would have touched a horizontal line drawn through the lower margin of the optic disc. It lies one optic disc's diameter external to the optic disc.



Right Eye.

The animal lived for twenty-nine days.

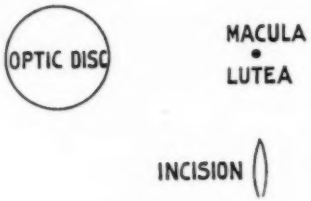
Microscopical examination of the right optic nerve.—Throughout the entire length of the nerve there is a well-marked degeneration situated ventrally. It is roughly triangular, its base being at the periphery.

In the anterior part of the chiasma a degeneration is present in the ventral part of the right side. The bulk of the degeneration retains this position and situation, but at the posterior part of the chiasma a few of the fibres have crossed.

Monkey 7 (Macacus Rhesus).

In the left eye a similar incision was made with a cataract needle in the same way as was done with the Graefe knife in the previous case.

Ophthalmoscopic appearance on withdrawal of needle.—A bright red area with well-defined margins was seen; its length was about half an optic disc's diameter. It lay one and a quarter discs' diameter below the yellow spot.



Left Eye.

The animal was killed after thirty-one days.

Microscopical examination of the left optic nerve.—There is found throughout the whole length of the nerve a degeneration with the following characters:—

It occupies a well-defined area in the ventral part of the right ventral quadrant. This area contains a small number of degenerated fibres; it lies chiefly at the periphery, and comes in contact with about one-twelfth of the circumference of the nerve. Some of the black points extend to half way between the central vessels and the edge of the nerve.

Chiasma.—In the front part of the chiasma the degeneration lies ventrally in the left half, and is close to the periphery. There is no degeneration in the right half at this level. Further back, the degeneration passes further outwards and is more diffuse. A few black points, however, are seen nearer the centre of the chiasma, and there are now a few in the right half of the chiasma which become more numerous in the sections further back.

Monkey 8 (Macacus Rhesus).

A lesion of the right retina was produced by a Graefe knife.

Ophthalmoscopic examination immediately before the death of the animal.—The lesion was seen at the inner part

of the fundus at a distance of one and a half optic discs' diameter from the inner edge of the optic disc. Its long axis was vertical and measured half an optic disc's diameter, its breadth was the sixth of its length. The lesicn was very white, and its outer margin was bordered with black pigment.

Right Eye.

Microscopical examination of right nerve.—There is a beautifully marked degeneration at the inner part of the nerve, throughout its whole length. Anteriorly it is wedge-shaped with its base at the periphery, posteriorly it is less triangular and forms a narrow band, lying close to the inner margin of the nerve.

Chiasma.—In its anterior part the degeneration lies wholly in the right half, close to the middle line, which is here distinguished by a septum. (Plate III.)

Further back the black points gradually cross to the other side in distinctly defined bands. (Plate IV.)

At the posterior part of the chiasma the degeneration has entirely crossed to the left side.

Monkey 9 (Bonnet Monkey).

A vertical incision was made at the outer part of the left eye near the equator. In this case the method of operation differed from that employed in previous cases. The lesion of the retina was produced by puncturing the coats of the eye with a Graefe knife at the site selected so that the vitreous had not to be traversed in making the lesion.

After death the globe was opened and a distinct linear lesion about 2 mm. long was found at its outer part in the equatorial region. The vitreous showed nothing abnormal.

The animal was killed thirty-five days after operation.

Fig. 3.

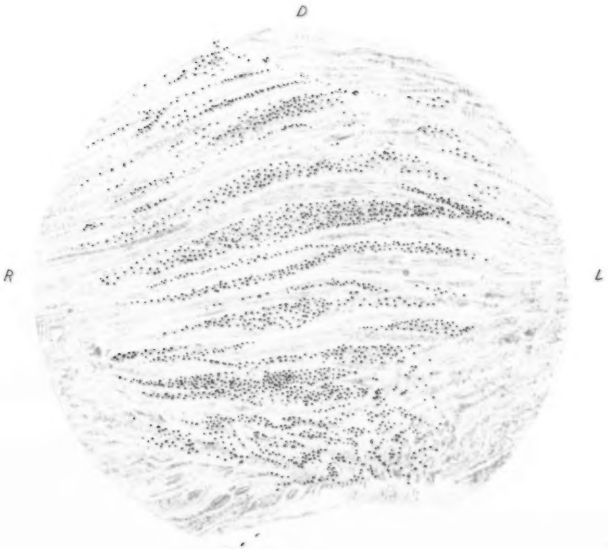


Fig. 4.

Bale & Danielsson Ltd. Lith.

Microscopical examination of the left nerve.—A degeneration is found in the outer dorsal part of the nerve throughout its whole length. The rest of the nerve is free from points of degeneration.

Chiasma.—The sections from the anterior part of the chiasma cut badly and could not be made use of. Those about the middle of the chiasma show that most of the degeneration lies in the left half near the lateral periphery, but there are also some black points in the ventral part of the right half.

Passing backwards the degeneration becomes less in the left side of the chiasma and approaches the middle line, whilst it becomes more marked at the ventral part of the right half of the chiasma. Definite bands of degenerated fibres are seen running close to the ventral periphery from the one side to the other.

At the most posterior part only a very few points of degeneration are left in the operated side, the bulk of them having crossed.

Group III.—Retinal Lesions at the Macular Region.

Monkey 10 (Macacus Rhesus).

The facts in regard to this experiment in so far as the nerve was concerned were published in our previous communication, but we may here briefly recapitulate them.

A lesion was made in the retina of the right eye between the optic disc and the yellow spot by means of a fine thermo-cautery. There was found at the anterior part of the nerve a well-defined area of degeneration, triangular in outline, with its base at the outer periphery, its apex close to the central vessels. The area occupied about one twelfth of the nerve-section. In the middle and posterior parts of the nerve the degeneration lay in the centre of the nerve, no part reaching the periphery.

The chiasma, which has since been examined, shows the degeneration on the same side as the lesion, quickly

reaching the dorsal periphery towards the middle line. The degeneration extends towards the opposite side along the dorsal periphery. (Plate V.) Near the centre of the chiasma about half the degenerated fibres have crossed, and at the posterior part nearly all have reached the other side, retaining, however, their dorsal situation.

Monkey 11 (Macacus Rhesus).

The lesion in this case was produced in the left eye by the thermo-cautery.

Examined ophthalmoscopically immediately after the operation the fundus showed a large hæmorrhage with well-defined margins extending from the optic disc to about one optic disc's diameter external to the yellow spot. Its breadth is equal to two or three discs' diameters. The right fundus was found to be normal.

The animal was killed by chloroform thirty-one days after the operation.

Microscopical appearance of left optic nerve.—In the anterior portion of the nerve at its outer side there is a well-marked degeneration, which is wedge-shaped, with the apex at the central vessels and with the base at the periphery.

Towards the middle of the nerve the degeneration comes to lie near the centre, but is more in the ventral than in the dorsal half of the sections. It maintains this position throughout the rest of the nerve.

Chiasma.—In the front part the degeneration occupies the same relative position as it did in the posterior part of the left nerve. Further back it passes towards the centre of the chiasma and becomes more dorsal. Although the coarser black points do not reach the dorsal margin numerous finer points are scattered in this region.

More posteriorly so many of the degenerated fibres have passed over that about equal numbers lie on both sides of the central part of the chiasma.

Just in front of the optic tracts the degeneration is found to have completely crossed to the right side.

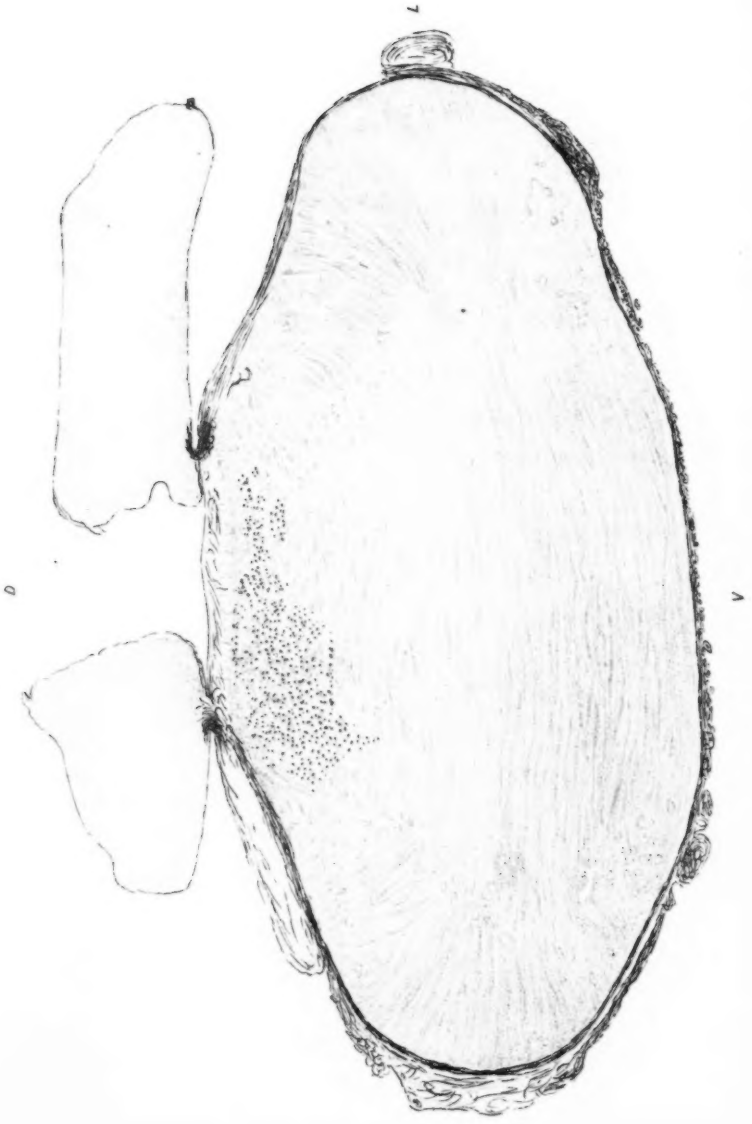


Fig. 5.

Bale & Danielsson Ltd, Lith.

Monkey 12.

In this experiment a well-defined lesion was produced between the yellow spot and the optic disc. The degeneration was found to occupy the same position in the nerve as in the two previous experiments. No other black points were found elsewhere in this nerve.

Owing to tilting of the chiasma during embedding, the examination was not quite so satisfactory as in the two previous macular cases, but all the facts observed tend to confirm the results obtained in these.

In connection with this group we would record the following case:—

Monkey 13.

The condition of the fundus described in this monkey was not the result of operation, but was observed during an ophthalmoscopic examination previous to an intended experiment. The animal was a male Macacus Rhesus.

The outer part of both optic discs was found to be markedly pale. The lamina oribrosa was conspicuous in this situation. The whole appearance presented a marked contrast to that of the discs of the monkeys which we had previously examined. The rest of the fundi, including both yellow spots, appeared normal, and the media were clear.

Microscopical examination of the optic nerves.—A tract of degeneration extends along the whole length of both optic nerves and through the chiasma to the optic tracts. The area occupied by this tract at the front of the nerves is triangular, with its base at the outer periphery, its apex at the central vessels. Further back in the nerves it becomes circular, and attains a central position, which it maintains as far as the chiasma. Anteriorly in the chiasma the degenerated fibres still occupy the central area of both halves. Soon after entering the chiasma they show a tendency to pass towards the dorsum, even before any crossing has taken place. (Plate VI.) The first crossing of these fibres occurs at the dorsal periphery (Plate VII.), and as the sections proceed backwards the crossing degenerated

fibres extend from the dorsal surface more and more ventrally until the affected area occupies the dorsal two-thirds of the central part of the chiasma. (Plate VIII.) The degeneration never reaches the ventral periphery.

The degeneration occupies about one-third of the sections in the nerve, and at least that proportion in the chiasma. The affected tract throughout its whole extent is very well defined, and can be easily followed. The structure of the degenerated tract consists largely of newly-formed fibrous tissue. In sections stained by Marchi's method the affected area contains a fair number of well-defined black points. No gross lesion could be found in the nervous system to account for this condition.

Microscopical examination of the nerves on the unoperated side.—A microscopical examination was made of all the nerves from the side of the unoperated eye. In every case small black points were observed. This was in accordance with our previous experience, which had been obtained for the most part from experiments on rabbits. Various hypotheses to account for these points were discussed in our previous paper.

With the view of attempting to elucidate the difficult question of the origin of these points we carried out a number of tests on the following lines :—

Sections from the front and back parts of the nerves were examined and compared with the view of finding whether the points were more numerous towards the eye-ball or towards the chiasma. The idea was that if they were due to myelin drops in process of transportation from the nerve, with the lesion to the nerve on the unoperated side, they would probably be more numerous posteriorly. No difference, however, could be ascertained between the two parts.

Comparisons were instituted between these nerves obtained from cases where there were marked lesions, such as division of the nerve, and those in which the lesions were very small. The object of this examination was to ascertain whether there was a correspondence between the number of black points in the nerve of the unoperated eye and the extent of the lesion on the operated side. If out of a series

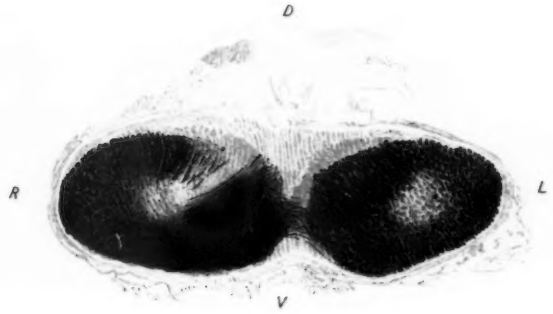


Fig. 6.

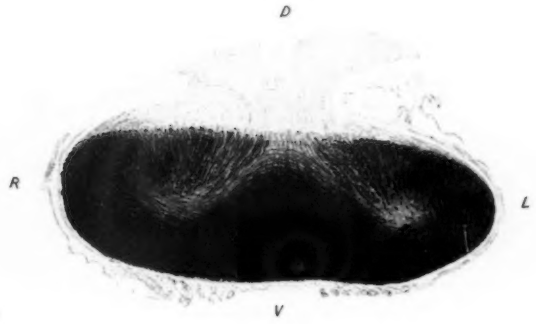


Fig. 7.

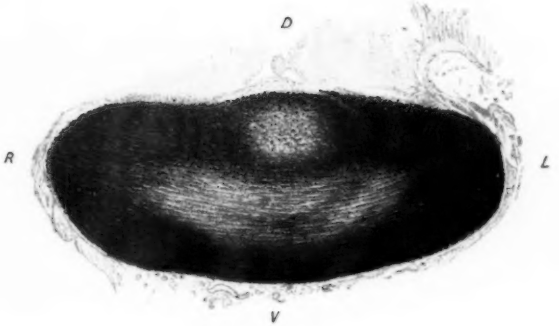


Fig. 8.

Bale & Danielsson Ltd. Lith.

of specimens which were unknown to the observer at the time, a selection was made of those containing most black points, the slides selected were generally, but not invariably, found to belong to the cases where the optic nerve had been divided.

The points are always distributed throughout the sections; we have never been able to determine the existence of definite groupings. The whole question is an extremely difficult and uncertain one, and though we have had more material at our disposal we are able to add nothing to our remarks in the *Ophthalmological Transactions*.[1] None of the optic nerves on the uninjured side in our cases show so many, or such large black points as are represented by Parsons in his paper. (Plate II.) In this connection it may be mentioned that Parsons employed Busch's Formol-Marchi method.

For a *résume* of the difficulties attending the use of Marchi's method we may refer to Spielmeyer's recent paper.[2]

Remarks and Conclusions.

Group I.

(Division of an optic nerve, five cases.)

Results obtained in the Optic Nerve.

The experiments in this group were made partly with the object of ascertaining whether any difference existed in the characters and extent of the degeneration in the parts of the nerve above and below the scar.

In three of the cases degeneration, as indicated by osmic acid staining, was most marked above the scar. In these cases, however, there were below the scar profound changes which involved largely the connective tissue framework and its relation to the nerve bundles. In this part of the nerves osmic acid staining was a much less marked feature than

[1] Ref. *loc. cit.*

[2] Walther Spielmeyer, "Die Fehlerquellen der Marchi'schen Methode," *Centralbl. f. Nervenheilkunde*, xxv., July 15, 1903.

in the part of the nerves above the scar. It appeared to us that a considerable amount of the difference in appearance above and below the scar was due to interference with the vascular supply, as was suggested by the ophthalmoscopic appearances, more especially in cases of Monkey 3 and Monkey 4.

In Case 1, which lived for only eight days after operation, the degeneration as evidenced by black points was extremely well marked below the incision, but scarcely at all above the incision.

Results obtained in the Chiasma.

(1) The degenerated fibres begin to cross over as soon as they enter the chiasma.

(2) The first crossing is seen to take place at the ventral periphery, due probably to the first junction of the optic nerves occurring ventrally. As the degenerated fibres proceed backwards they invade more and more of the chiasma from the ventral to the dorsal surface, till ultimately they are distributed over the whole central part of the chiasma.

(3) Only a few, if any, of the crossed fibres appear to approach the lateral periphery of the half of the chiasma corresponding to the unoperated nerve, whereas on the corresponding area of the opposite side the degeneration is dense.

(4) It could not be determined that the crossed fibres at the posterior part of the chiasma were more numerous than the uncrossed.

(5) A very definite tract of crossed fibres lies at the ventral part of the chiasma posteriorly.

Wieting [1] examined the optic nerves, chiasma, and tracts of three cases in which there was complete atrophy of one of the optic nerves. He stained the sections by Weigert-Pal's method and found partial crossing of the fibres in the chiasma. The crossed fibres occupied a ventral median area which increased in size towards the posterior part of the chiasma.

[1] Wieting, "Zur Anatomie des menschlichen Chiasma," *Arch. f. Ophth.*, 1898, Bd. xlv., Abt. i., S. 75.

Dimmer[1] investigated two cases by Marchi's method. First case:—Enucleation of an eye, whose optic nerve was normal, six weeks before death. Second case:—Complete amaurosis of one eye and temporal hemianopia of the other, brought about by a tubercular growth of the chiasma. He found the crossed bundle lying chiefly in the ventral median part of the chiasma. It may be noted that the sections of the chiasma of his first case, figured in his paper, present a striking resemblance to the chiasma sections in our Group I.

Group II.

(Retinal lesions at a distance from the macula, four cases.)

Results obtained in the Optic Nerve.

The results in all the experiments as regards the degeneration are in accordance with those obtained by us in rabbits. The degeneration lies in the part of the sectional area of the nerve corresponding to the site of lesion in the retina. Experiments 6 and 7 are almost identical. In both the retinal lesions were below the macular region. The degeneration occupies a ventral position throughout the nerve and chiasma, and in both cases there is a slight crossing of the degenerated fibres.

Results obtained in the Chiasma.

(1) A ventral degeneration in the nerve (Cases 6 and 7) maintains a ventral position in the chiasma. A very few degenerated fibres cross to the other side.

(2) A lesion at the inner part of the fundus (Case 8) is followed by a degeneration at the inner part of the nerve which, in the front part of the chiasma, lies on the same side, but ultimately crosses completely to the opposite side.

(3) A very peripheral lesion in the outer part of the fundus (Case 9) is followed by a degeneration in the outer dorsal part of the nerve. In the chiasma the degeneration lies on the same side close to the lateral periphery,

[1] Dimmer, "Zur Lehre von den Sehnervenbahnen," *Arch. f. Ophthal.* 1899, Bd. xlviii., Abt. iii., S. 473.

ultimately crossing and occupying the ventral periphery of the opposite side, only a few fibres remaining uncrossed at the posterior part of the chiasma. This experiment makes it probable that in this particular monkey (a specimen of the Bonnet Monkey), the great majority of the fibres cross in the chiasma. It is possible that this indicates a reversion to the lower or more primitive type of chiasma. Such a view is suggested by Schäfer[1] to account for certain reported human cases which can be explained only on the hypothesis of complete crossing of the fibres. It may be noted, however, that Parsons (BRAIN, Autumn Number, 1902, part xcix.) in two cases of temporal lesion, one in a Bonnet Monkey, the other in a Macacus Rhesus, found that a certain number of degenerated fibres were present in the opposite tract.

GROUP III.

(Retinal lesions at the macular region, three cases.)

Before giving a summary of our conclusions as to the course of the macular fibres in the optic nerve and chiasma, based on Case 13 and on the experimental cases in this group, it may be desirable to give a brief statement of the most important work on the subject of the course of the macular fibres.

Henschen[2] studied clinically and pathologically a large amount of human material and compared his results with the cases recorded in the bibliography, and gives his conclusions regarding the localisation of the bundles in the optic nerve, chiasma, and tract. He states that the bundle from the macula lutea on each side runs in the central part of the optic nerve, and maintains its central position in the optic chiasma and in the optic tract.

Vossius,[3] in a case of symmetrical central scotoma in

[1] Schäfer, "Quain's Anatomy," vol. iii., pt. i., p. 118.

[2] Henschen, "Klinische und anatomische Beiträge zur Pathologie des Gehirns," Zweiter Theil, Upsala (1892), S. 217, ref. Barker, "The Nervous System," p. 793.

[3] Vossius, *Arch. für Ophthalm.*, xxviii., 3, 201.

man, found that the macular fibres lie close under the dorsal margin of the chiasma. It will be noted that Case 13 reported by us corresponds very closely with Vossius' description and drawings.

Bunge[1] found that the papillo-macular bundle maintained a dorsal position throughout the chiasma in man.

As having a practical bearing on the question of the exact situation of the macular fibres in the chiasma should be mentioned such cases as those recorded by Nettleship[2] of central amblyopia from tumour at the chiasma.

Results obtained in the Optic Nerve.

In all cases the degeneration is situated on the outer side of the nerve in front, while further back it reaches a central position which it maintains as far as the chiasma. The shape of the area in the anterior part of the nerve is triangular, the base being at the periphery, the apex at the central vessels.

Results obtained in the Chiasma.

The course of the macular fibres in the chiasma is as follows :—

Anteriorly in the chiasma they occupy the central area of both halves. Soon after entering the chiasma they pass towards the dorsum before any crossing has occurred. The crossing takes place first at the dorsal periphery, at some distance behind the front of the chiasma. On proceeding backwards, though still partly remaining dorsal, it spreads from the dorsal surface in a ventral direction. The macular fibres never reach the ventral periphery.

As regards the question whether the macular fibres cross wholly or partially in the chiasma it is impossible from our experimental cases to make a definite statement, owing to the difficulty that exists in determining whether the wound was limited to the macular fibres or, on the other

[1] Bunge, "Ueber Gesichtfeld," p. 21.
[2] Nettleship, *Trans. Ophth. Soc.*, 1897, vol. xvii.

hand, whether it had damaged only a portion of these; while in Case 11 the decussation was complete, in Case 10 there were uncrossed fibres, even at the most posterior part of the chiasma. No help could be obtained in elucidating this point from Monkey 13, since in that case both optic nerves were affected.

With the view of studying by means of Marchi's method the degeneration resulting from tobacco poisoning we endeavoured to obtain this condition in two monkeys by feeding them with tobacco. The treatment lasted for six months in one case and for about one year in the other.

The optic nerves on examination showed in both cases a slight diffuse degeneration. This agrees with what was found by Barrett in his researches on the tobacco amblyopia of the horse in Australia.[1]

De Schweinitz[2] failed to establish toxic (alcoholic) amaurosis in monkeys and dogs, probably, he adds, because the drug was not continued for a sufficiently long period.

Birch-Hirschfeld[3] gave methyl-alcohol to seven rabbits and three fowls. In one of the rabbits a wedge-shaped temporal area was found immediately behind the globe, with partial destruction of the medullary sheaths. In the other rabbits and fowls there was no degeneration in the optic nerves. The same author[4] found no changes in the optic nerves of four dogs to which methyl-alcohol had been given. Only one of three monkeys treated in the same way showed changes in the optic nerves, and the degeneration was limited to the anterior part of the nerves, it lay on the outer part and formed a wedge-shaped area. The rest of the nerves, the chiasma and tracts showed no sign of degeneration.

The pathological condition present in Monkey 13 corresponds more nearly to the ordinary type of lesion found

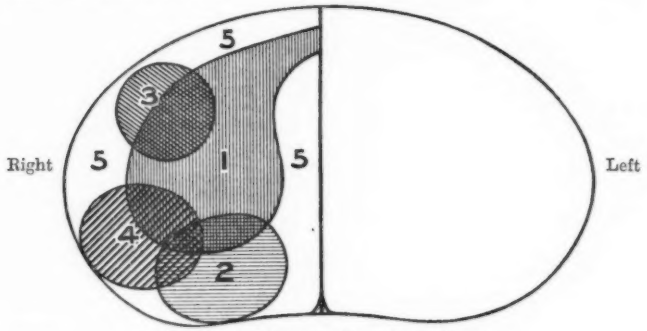
[1] "System of Diseases of the Eye," Norris and Oliver, vol. iv., p. 817.

[2] De Schweinitz, "System of Diseases of the Eye," Norris and Oliver, vol. iv., p. 809.

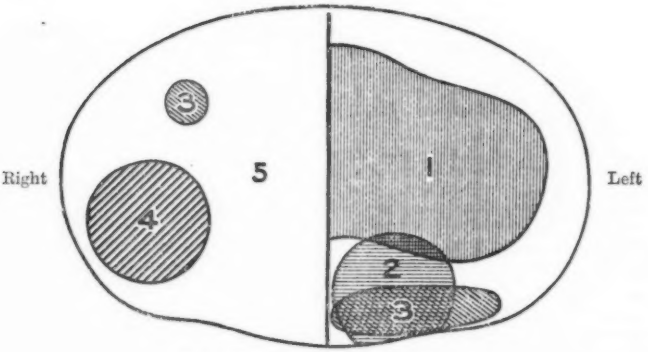
[3] Birch-Hirschfeld, "Experimental Untersuchungen über die Pathogenese der Methyl-alcoholamblyopie," *Arch. für Ophthalmologie*, 1901, zweites heft 358.

[4] Birch-Hirschfeld, "Weiterer Beitrag zur Pathogenese der Alcohol-amblyopia," *Arch. für Ophthalmologie*, 1902, Band liv., erstes Heft 68.

in the optic nerves of man as a result of tobacco poisoning.[1] We have endeavoured to get the previous history of this animal with reference to a tobacco habit, but have not succeeded in doing so.



Front of chiasma. Seen from front.



Back of chiasma. Seen from front.

Scheme of the situation of optic nerve-fibres in the chiasma in so far as they can be based on the results of the foregoing experiments.

(1) Macular fibres—Experiments 12, 10, 5, and Case 13.
(2) Bundle of crossed fibres—Experiments 1, 2, 3, 4, 5, 8.
(3) Bundles of crossed and uncrossed fibres—Experiment 9.
(4) Bundle of uncrossed fibres—Experiments 6 and 7.
(5) A large number of uncrossed fibres occupying the clear area, as shown by Experiments 1, 2, 3, 4, 5.

[1] Samelsohn, "Graefe-Saemisch Handbuch der gesammten Augenheilkunde," Auflage i., Bd. v., S. 834, 1877; Nettleship, *Trans. Ophth. Soc.*, vol. i., 1882; Vossius, *Archiv. für Ophthalmologie*, Bd. xxviii., 3, 1883; Uhthoff, *Graefe's Archiv.*, Bd. xxxii., 3, S. 95.

Description of Plates.

Fig. 1 illustrates the microscopical appearances of a coronal section through the most anterior part of a monkey's chiasma. The left optic nerve had been divided during life. The section was prepared by Marchi's method. The darkly-stained left half of the chiasma consists of degenerated fibres.

Fig. 2 illustrates the microscopical appearance of a coronal section from the anterior part of the same chiasma as is shown in figure 1, but further back in the chiasma. The section was prepared by Marchi's method. Degenerated fibres are seen crossing from the left half to the right half of the chiasma. Most of the crossing fibres are in the ventral part of the chiasma and are arranged in transverse bands.

Fig. 3 illustrates the microscopical appearance of a portion of a coronal section from the anterior part of a monkey's chiasma. A lesion at the inner part of the right retina had been made during life. The section was prepared by Marchi's method. The whole of the darkly-stained degenerated area is shown in the plate, and is limited to the right half of the chiasma. A small septum is situated at the mid line ventrally.

Fig. 4 illustrates the microscopical appearances of a coronal section from further back in the same chiasma as is shown in figure 3. Only a portion from the centre of the section is illustrated. This includes the whole of the darkly stained parts. The degenerated fibres are crossing in transverse bands from the right half to the left half of the chiasma.

Fig. 5 illustrates the microscopical appearances of a coronal section from a monkey's chiasma. The papillo-macular fibres of the right eye had been injured during life. The section was prepared by Marchi's method. Degenerated fibres are passing at the dorsal periphery from the right half to the left half of the chiasma. D = dorsal, V = ventral, R = right, L = left.

Figs. 6, 7 and 8 illustrate the microscopical appearances of coronal sections through the chiasma of a monkey. The sections were stained by Marchi's method. No. 6 is most anterior and No. 8 most posterior. The chiasma was from a monkey whose optic nerves showed degeneration corresponding exactly in situation to that found in tobacco amblyopia in man and in macular lesions in monkeys. The degeneration had not been produced by experiment. In figure 6 degeneration is seen at the centre of each half—on the right side the degeneration is seen to pass dorsally, and this is still more clearly shown on both sides in figure 7. In figure 8 the degeneration, although still partly remaining dorsal, has spread in a ventral direction, but does not reach the ventral periphery.

POST-DIPHTHERITIC CHRONIC BULBAR PARALYSIS, AND ITS DISTINCTION FROM MYASTHENIA.

BY WILFRED HARRIS, M.D., M.R.C.P.,

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CHRONIC bulbar paralysis occurring as a sequel of diphtheria is a rare event, and I can find only four instances in the literature. Two cases are described by Gowers (18), in both of which primary optic atrophy and external and internal ophthalmoplegia developed slowly some time after the diphtheria, in one of them six months afterwards. In his other case there was additional weakness of the lips, tongue, and pharynx. Stadthagen (41) in 1884 described a case of a boy, aged 11, who at the age of 4 had suffered with diphtheria, followed four weeks later by paralysis of the palate, nasal speech, and regurgitation of food, and later by gradual weakness of the arms and legs. When aged 11 he still presented the symptoms of chronic bulbar palsy, paralysis of the lips, tongue, and palate, with slight weakness of the lower face. Collins (10) in commenting on this case considered it to be a case of associated bulbar neuritis, rather than a chronic nuclear lesion, but Gowers (19), when referring to Stadthagen's case, says: "In extremely rare cases some part, as the palate, has not recovered, and other paralyses have developed and persisted, apparently due to chronic disseminated myelitis." Dundas Grant (12), in 1892, published a case of persistent paralysis of the muscles of mastication after diphtheria.

In the two cases which I am now publishing, one of which I showed at the Harveian Society a year ago, and also at the Clinical Society in January, 1904, the similarity of the

mode of onset of the symptoms and the distribution of the paralysis have led me to regard them as a distinct type as yet unrecorded. In both the cases, after an attack of ulcerated sore throat, there developed a few weeks later paralysis of the palate, followed soon afterwards by weakness of the tongue and lips and inability to close the eyes. In each case there was profound lasting paralysis of the sphincters of the eyes and lips, with complete escape of the remaining facial muscles, with the exception of the frontalis, which appeared to be slightly weak in both. In one case the symptoms lasted six years, followed by complete recovery; in the other, a more severe case, with more marked atrophy of the tongue and facial muscles, the symptoms remain without any improvement after the lapse of four years.

Several cases have been published of chronic bulbar palsy, with involvement of the upper facial muscles, by Wachsmuth (43), Remak (36), Eisenlohr (13), Oppenheim (33), Brissaud and Marie (6), and others.

A progressive juvenile form of bulbar palsy was described by Fazio in 1892 (14) in a girl aged $4\frac{1}{2}$, whose mother was also suffering from a most complete form of bulbar paralysis, with involvement of the upper facial muscles. The child had almost complete paralysis of both the upper and lower facial muscles, with defective articulation, difficulty in breathing and in swallowing, with atrophy of the tongue. There was paralysis of the left vocal cord, less marked of the right. The limbs were normal. Complete reaction of degeneration was present in the facial muscles. An interesting point, probably bearing on the cause of the child's condition, was the fact that it was born five months before the development of symptoms in the mother. Bernhardt had previously (2) published a case of chronic bulbar palsy in a child, but without involvement of the upper facial muscles, and a similar case was also published by Hoffman (22) of chronic progressive bulbar palsy in a boy, aged 11, which commenced with salivation, followed five weeks later by difficulty in swallowing, with nasal regurgitation. Paralysis of the lips followed, and of the middle and lower facial muscles, but with escape of the upper facial muscles. In addition

there was paralysis of the abductors of the vocal cords, and of the left half of the soft palate. There was also atrophy of the tongue and some of the trunk muscles. Reaction of degeneration was present in the lips. Hoffmann insists that diphtheria can be excluded as the cause, but Londe (28), in referring to the case, draws attention to the commencement of the paralysis with palatal palsy.

Charcot (9) in 1893 described two cases of the progressive juvenile type of chronic bulbar palsy in two brothers, aged 5 and 9. Londe, who reported the cases for Charcot, published them at greater length (28), and Brissaud (5) had published the case of the elder brother as a case of probable bulbar tumour. Londe states that the symptoms commenced in the elder boy, when aged $8\frac{1}{2}$, with inability to close the eyes completely. This was followed by defect in articulation and paralysis of the tongue and of the lower facial muscles, including the lips. The forehead was immobile, with paralysis of the frontales and corrugators. Nasal regurgitation, some abductor paralysis of the vocal cords, and slight left ptosis were present. There was no true reaction of degeneration, but diminution to both forms of current was found in the paralysed muscles. The knee jerks were brisk, and there was no weakness of the limbs or sphincters. No variability in the symptoms was noticed. The paralysis in the younger brother was limited to the upper face, there being paralysis of the orbicularis palpebrarum and of the frontalis and corrugator, but there was no difficulty in swallowing or of articulation, and scarcely any weakness of the tongue or lips. Charcot considered the cases to be progressive bulbar nuclear degeneration, and drew attention to the resemblance to the myopathic facies, due to the symmetrical involvement of the sphincters of the eyes and lips. Myasthenia can, I think, be excluded in these two cases of Londe's, on account of the progressive weakness without variability of the symptoms, and the alteration in electrical reactions and the fact of its occurrence in two brothers, suggesting a familial disease. It is, however, apparent that Londe was unacquainted with myasthenia, as he refers as a similar case to a case of Remak's (37),

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which is a clear case of myasthenia. This was the case of a girl, aged 12, in whom the symptoms followed otitis media after influenza, and ran a fatal course in four months. The symptoms commenced in this case with inability to close the eyes, and defect of articulation. Later difficulty in swallowing, paresis of the facial muscles, of the tongue and palate, and lips. Frequent attacks of dyspnœa from weakness of the respiratory muscles, ending in death from respiratory failure.

Although my two cases bear a certain superficial resemblance to myasthenia, it is evident on studying them carefully that they must be considered as belonging to a separate class. I shall return to the differential diagnosis from myasthenia after detailing the cases in full:—

Case 1.—M. L., a young woman, aged 25. Four years ago, in November, 1899, she suffered from a slight attack o ulcerated sore throat, which was treated, but no suggestion of diphtheria was made. Six weeks later she noticed difficulty in swallowing, and nasal regurgitation, for which she went to see Dr. Ferris, of Uxbridge, who found that the soft palate was paralysed, and told her that she must have passed through an attack of diphtheria. Dr. Ferris in August, 1902, sent her to St. Mary's Hospital, where I saw her, and he has kindly given me some notes on the early history of the case. Her voice was altered from the first, and soon afterwards her lips became affected, so that she could no longer purse up her mouth, or whistle. It was not until January, 1901, that much difficulty in articulation was noticed, and about this time she found that her eyelids were getting weak, and that she could no longer close her eyes properly. There was never any ptosis or weakness of the jaws or limbs, nor numbness, or any affection of vision. From that time onwards she became slowly slightly worse, the weakness of the eyelids and lips increasing, and the articulation becoming more indistinct, but with occasional slight temporary improvement of the articulation and of swallowing. When I first saw her on August 5, 1902, her condition was much as it is now, with the exception that weakness of the abductors and internal tensors of the vocal cords has since developed, and the voice is more nasal, owing to the paralysis of the soft palate having become complete, there having been slight movement of the velum on phonation a year ago. She has quite recently married.

FIG. 1.

Case 1.—M. L. This photograph shows the absence of ptosis, and the normal power of the muscles at the angle of the mouth, in the act of showing the teeth.

FIG. 2.

Case 1.—M. L. This photograph, taken immediately after the former, shows the complete paralysis of the sphincters of the eyes and lips, in the endeavour to close the eyes and to purse up the lips.

Present state.—There is almost complete paralysis of the orbicularis oris and of the orbicularis palpebrarum on each side, with inability to purse up the lips, or to close the eyes completely. The facies resembles that seen in the myopathy of Landouzy-Dejerine. The remainder of the facial muscles are normal, with the exception of slight weakness of the frontalis, only the sphincters of the eyes and lips being involved (see figs. 1 and 2). The articulation is nasal, and there is complete paralysis of the soft palate, which hangs motionless on phonation, with loss also of the reflex. There is also considerable wasting and paralysis of the tongue, which cannot be protruded properly. There is also weakness of the constrictors of the pharynx, with bilateral weakness of the abductors and internal tensors of the vocal cords, which during phonation do not lie properly adjacent, but leave an elliptical space between. Occasionally she has been unable to open the eyes on waking, but there is never any ptosis. There is no weakness in chewing, or of the arms or legs. She says she tires rather easily, but no loss of power is to be detected in the muscles of the limbs after repeated exertion more than is to be expected in a slightly built woman. All the deep reflexes are brisk, but not excessively so. The plantar reflex is flexor. Before I saw her she had been treated in another hospital by injections of strychnine, which, she thinks, made her worse. For about a year I have treated the palate and pharynx, and facial muscles with galvanism, at first twice a week, afterwards once a week, but without improvement.

Electrical reactions.—Very diminished and sluggish contractions to faradism in the sphincters of the eyes and lips, with stronger and markedly sluggish contractions to galvanism, KCC > ACC. The soft palate reacts only to galvanism, with a sluggish contraction, KCC > ACC. The tongue reacts more briskly both to faradism and to galvanism, but much less so than normal. There is no exhaustion to faradism of the facial or jaw muscles or biceps after tetanisation for two minutes, therefore the myasthenic reaction is absent, and the reaction of degeneration is present, best marked in the soft palate and in the orbicularis palpebrarum. Probably at the commencement there was even better marked R.D. in the palate, perhaps with the polar change ACC > KCC, but, as usually happens in a case of long-standing paralysis, the kathodal contraction has regained its superiority. The sensation is perfectly good on the face and tongue, but there appears to be considerable diminution of sensation on the pharynx and palate.

NUCLEAR SUPPLY OF THE UPPER FACIAL MUSCLES.

The difficulty in opening the eyes in the morning, which she has noticed on several occasions, having to push up her upper eyelids three or four times before she can keep her eyes open, is not to be confounded with real weakness of the levator palpebræ, which, if present, would certainly be most manifest at the close of the day instead of at the commencement, especially were it a case of myasthenia. It is, no doubt, to be explained, as Brissaud has stated, as being due, not to any weakness of the levator palpebræ, but to the paralysis of its antagonist, the orbicularis palpebrarum. The difficulty then of raising the eyelid may be looked on as comparable to an ataxy; the normal muscular tension of the orbicularis not being opposed to the action of the levator, and its appearance only in the morning in this patient on waking, is thus explicable by the fact of the levator palpebræ having been disused for several hours. This point is of some importance, partly because ptosis is such a common symptom in myasthenia, and also for the reason that if the temporary difficulty in raising the eyelids were ascribed to weakness of the levator palpebræ on each side, it would indicate that the third nucleus was partially involved, and on account of the upper facial weakness present in this case, would thus be of some support to the view originated by Mendel (30) in 1887, after extirpating the orbicularis and frontalis in dogs, that the upper facial muscles were represented in the posterior portion of the third nucleus, and not in the facial nucleus proper. That this view is probably incorrect has been shown by Schwabe (38), who found by the use of Nissl's method after section of the facial nerve that extensive cell changes took place in the facial nucleus, but not a single cell appeared altered in the third nucleus. Marinesco (29) has also performed a similar experiment in dogs, dividing the branch of the facial nerve to the three muscles, orbicularis palpebrarum, frontalis and corrugator, the facial *supérieur*, and examining the nuclei by Nissl's method fifteen days later. He divides the facial nucleus into three portions, upper, middle,

and lower, and found the *réaction à distance*, or chromatolysis, in the lower cell-group and lower portion of the middle group. He concludes from this that this portion of the facial nucleus is the nucleus for the upper facial muscles, as he found all the cells of the third nucleus normal. Van Gehuchten (47) has confirmed Marinesco's work in the rabbit, and he divides the facial nucleus into four cell-groups, three anterior, parallel to each other, and one posterior. The latter he concludes to be the nucleus of the upper facial muscles, as he found marked chromatolysis in the cells of this group alone, after dividing the facial *supérieur*, the cells of the third nucleus showing no changes by Nissl's method. On the other hand, a considerable number of cases have been published of ophthalmoplegia with weakness of the orbicularis palpebrarum or frontalis, or of both, by Hughlings-Jackson (23), Smith (40), Hanke (21), Birdsall (3), and others. With regard to the weakness of the orbicularis palpebrarum in these cases a possible source of fallacy has, I think, been overlooked, namely, an apparent weakness only of the orbicularis due to the paralysis of its antagonist, the levator palpebræ, in the cases of ophthalmoplegia with ptosis. This apparent weakness is exactly comparable to the apparent weakness of the levator palpebræ in cases of palsy of the orbicularis, its antagonist already referred to above as explained by Brissaud. An instance illustrating this point has lately come under my own observation—a woman with well-marked signs of tabes dorsalis, with complete external ophthalmoplegia and marked ptosis. When told to screw up her eyes tight she closed them perfectly, but the eyes could be easily opened by the fingers without using any force. At first sight, therefore, the orbicularis appeared to be weak, but, on telling the woman to screw the eyes up again, and urging her several times to screw them as tightly as possible, it was found that after three or four trials she could screw up the eyes as tightly as a normal person, but she always required considerable urging before this was accomplished. Since she was a tabetic, there was an additional reason for apparent weakness of the muscle, due to

ataxy, from sensory paralysis within the muscle, in addition to the paralysis of its antagonist.

Tooth and Turner (42) describe a case of bulbar paralysis with involvement of the lower facial muscles, in which, at the microscopical examination, they found complete degeneration of the facial nucleus, arguing therefrom that the upper facial muscles derive their nervous supply from the third nucleus through the posterior longitudinal bundle. There is, however, no anatomical basis for this view, beyond Mendel's original results, in which he stated that after extirpation of the orbicularis palpebrarum and frontalis muscles in dogs, he found the nerve-cells in the posterior portion of the third nucleus on the side operated on smaller and less numerous than on the other side, while the facial nucleus appeared normal. This was before the days of Nissl's method, and similar experiments done by this method have failed to substantiate Mendel's contention. Cassirer and Schiff (8), moreover, describe a case of complete degeneration of the oculo-motor nucleus, including its posterior portion, in which the upper facial muscles were normal. Siemerling and Boedeker (39) also state that they have found no evidence in support of the view that the upper facial muscles are supplied from the third nucleus.

Wilbrand and Saenger (45), who give a good abstract of most of the literature on the subject, declare their adherence to the view of Cassirer and Schiff, and of Siemerling and Boedeker, that the third nucleus has no connection with the upper facial muscles, and they further cite as evidence against Mendel's view the common overaction of the frontalis in nuclear ophthalmoplegia with ptosis. The question is thus still somewhat unsettled, and must be decided in the future by instances of third nuclear palsy (with autopsy) with uninjured facial nucleus, or by Nissl-stained sections of the nuclei in cases in which the upper facial nerve has been damaged about a fortnight before death. The numerous instances of pontine tumour or hæmorrhage, with paralysis of the whole face, upper and lower, are of no value, since the descending fibres from the third nucleus, if present, would be damaged on their way to join the facial nerve. Similarly

cases of acute degeneration of the seventh nucleus in polioencephalitis inferior are of little help, for the same reason of possible destruction of the descending fibres.

Of similar interest is the question of the nuclear origin of the fibres for the orbicularis oris, which it has been suggested indeed, with no want of dogmatic assertion on the part of some teachers, derive their nuclear supply from the hypoglossal nucleus. Gowers (20) leaves the question quite open, while showing the possibility of some of the facial fibres descending to, and perhaps arising from, the hypoglossal nucleus. Experiments of extirpation of the orbicularis oris with subsequent examination of the nuclei by Nissl's method may perhaps clear up this point, but nothing positive is yet known.

The following case of bulbar palsy following diphtheria is of great interest owing to its close resemblance to Case 1 in the distribution of the paralysis, and also to the fact that after the symptoms had persisted for six years, rapid recovery took place. The girl was ten years ago in the Queen Square Hospital under the care of Dr. Hughlings Jackson, for whom I was then clerking, and who has kindly given me permission to make use of the notes of the case.

Case 2.—L. C., aged 19 years, an unmarried girl, had diphtheria in the Summer of 1892. Three weeks later she had a good deal of difficulty in swallowing, with nasal regurgitation of liquids. Shortly after this, she noticed her tongue was clumsy and useless in eating. This was followed by diplopia, and soon by inability to close the eyes. There was slight trouble with the fingers, being unable to button her frocks, but there was no weakness of the legs. No family history of nervous disease. The diplopia soon improved, but the condition of the lips, eyelids, tongue, and palate remained unaltered, and she was admitted under the care of Dr. Hughlings Jackson on November 29, 1893, eighteen months after the attack of diphtheria. She still complained of slight diplopia, but there was no apparent ocular weakness. Pupils normal, and no ptosis. There was considerable weakness of the orbicularis palpebrarum, so that she could only just close her eyes, but very inefficiently. There was marked weakness of the orbicularis oris, so that she could not purse up her lips or whistle, or suck things. She could wrinkle the forehead,

and frown, but only badly, while all the remainder of the facial muscles escaped completely. The soft palate was completely paralysed, and the voice nasal. The reflex of the palate was also lost, but its sensation was good. The tongue was protruded straight, but she still complained it was clumsy. There was no wasting of the tongue. The laryngeal muscles were normal. No weakness of the jaw. After five months she had improved somewhat. The diplopia had disappeared, and there was no longer any nasal regurgitation, and she was now able to curl her tongue well, but the paralysis of the sphincters of the eyes and lips remained as before, and the palate also remained paralysed, and deglutition was performed with an obvious effort.

The electrical reactions on admission showed diminution to faradism in the orbicularis oris and orbicularis palpebrarum as well as in the frontalis and corrugator supercilii, while the remainder of the facial muscles reacted normally. On April 7, 1894, the reactions are noted as follows: "The orbicularis palpebrarum reacts readily to a very weak current, as readily as the levator anguli oris, though the weakness of the eyelid continues as before. The orbicularis oris reacts to the same current, but more sluggishly. All the lingual muscles react smartly on faradic stimulation of the nerve at the angle of the jaw, a very weak current was enough for the frontalis, while a strong, painful current was necessary before the sphincters of the eyes or lips would contract." After leaving the hospital in August, she attended the hospital for about three years as an out-patient for electrical treatment, and married in September, 1897, but there was little or no alteration until a year after her marriage, when she noticed rapid improvement soon after her first child was born. She has remained perfectly well for the last five years, after suffering with the bulbar symptoms for six years. I saw her in April, 1903, and found that she had recovered perfectly. All the movements of the eyes and face were normal, and the electrical reactions of all the facial muscles were normal and brisk. The palate had also completely recovered.

The course of this case, following diphtheria and recovering after pregnancy, recalls one of Saenger's cases of myasthenia (48). His was the case of a young married woman, aged 22, who suffered from ulcerated sore throat, and four weeks later developed difficulty in swallowing, diplopia, ptosis, ophthalmoplegia, paralysis of the palate and of the lower face, with weakness of the extremities and neck

muscles. There was no myasthenic reaction or reaction of degeneration. Campbell and Bramwell mention in their abstract that she began to improve in a fortnight, and subsequently, during her first pregnancy, recovered completely, but I can find no mention of the sequel of the case in Saenger's own description of the case (48).

The resemblance in type of my two cases is remarkable. In both there was complete paralysis of the palate, almost complete paralysis of the sphincters of the eyes and lips, with slight weakness of the frontalis, and complete escape of the muscles of expression round the mouth. In each the tongue was partially paralysed, and there was considerable difficulty in swallowing. In both the symptoms commenced with paralysis of the palate, in one three weeks, in the other six weeks, after an illness with ulcerated sore throat, which was recognised as diphtheria in one of them. There is no bacteriological evidence of diphtheria in either, even if that were considered conclusive, but the fact that paralysis of the palate, with nasal regurgitation, supervened a few weeks after an attack of ulcerated sore throat would be considered at the time, at all events, as conclusive for diphtheria, I imagine, by anyone. Because the after sequence of events was unlike the usual course of post-diphtherial paralysis is insufficient reason, in my opinion, for disregarding the previous diagnosis of diphtheria. In some respects the cases resemble myasthenia, or asthenic bulbar paralysis. The paralysis of the sphincters of the eyes and lips, with weakness of the frontalis and escape of the other facial muscles, is not uncommon in myasthenia, as is also weakness of the tongue and palsy of the palate, with difficulty in swallowing. Some of the most constant and typical symptoms of myasthenia are, however, conspicuous by their absence in both of these cases. Such are ptosis, weakness of the jaw muscles and of mastication, ophthalmoplegia, weakness of the limb muscles and of the neck, attacks of dyspnœa, and especially the variability in the symptoms, being worse after exertion and in the evening, with the myasthenic reaction in some of the affected muscles. All these signs are absent in my two cases, the symptoms being

remarkably constant, with no more than the slight variations that may be met with in any case of chronic nervous disease.

Of the 114 cases of myasthenia tabulated from the literature by Hun (24), weakness of the extremities was a symptom in every case but one, and that a doubtful case. Ocular paralysis was found in all but four cases, two of which were doubtful. Ptosis was present in 85 per cent. of the cases. Of the fifty-six definite cases of myasthenia abstracted by Campbell and Bramwell (7), ptosis and weakness of the jaw muscles were present in 80 per cent. and 82 per cent. respectively. Of the eleven cases of this series in which ptosis was not present, seven had weakness of the jaw muscles, and five suffered from attacks of dyspnœa. In every case, except Case 47, there was present one of the three symptoms—ptosis, weakness of the jaws, or attacks of dyspnœa—while in Case 47 there was weakness of the arms and legs, with the myasthenic reaction. Even the so-called myasthenic reaction, or rapid exhaustion of reaction to faradism of the muscle, has been shown to occur in numerous conditions besides myasthenia, such as hemiplegia, cerebellar tumour, neurasthenia and muscular dystrophies. It was first described as the *Reaction der Erschöpfbarkeit* by Benedikt in 1868 (46), twenty-seven years before Jolly (25) described it in myasthenia, and it has been similarly described in other forms of nervous diseases by Mosso (32), Kollarits (26), Flora (16) and Feinberg (15). On the other hand, this form of electrical reaction has been found absent in undoubted cases of myasthenia by Saenger (48), Sinkler (49), Punton (50) and Feinberg (51).

Of my two cases the second, perhaps, resembles myasthenia more than the other, in the absence of muscular atrophy, and of distinct reaction of degeneration, and in the presence of diplopia, with eventual complete recovery. Since, however, diplopia is such a common transient event in post-diphtheritic paralysis, it is not strange that it should form part of the symptom-complex of the more permanent type of bulbar lesion following that disease. In my first case the distinction from myasthenia may be summarised as follows: Little or no variability of the symptoms; absence of

any weakness of the jaw muscles, or of the neck or limbs; absence of the myasthenic reaction in any of the facial or arm muscles; absence of any ptosis or ocular palsy or diplopia; absence of any dyspnœic or choking attacks, or attacks of palpitation; the presence of atrophy of the tongue and lips; reaction of degeneration in the palate, orbicularis palpebrarum and orbicularis oris.

The two cases must be classed together, and it is evident that the first case is a more severe one than the second, though the lesion must be considered to be of the same type in both. What then is the lesion? In myasthenia, the bulbar nuclei, nerves and muscles have been found many times to be perfectly normal, and Campbell and Bramwell suggest that the pathological process is a diminution of functional activity of the peripheral nerve-fibres of the bulbar nerves, due to some circulating microbic toxin. More recently, however, Weigert (44) has found in a case of myasthenia lymphoid infiltration of the muscles, secondary to a lympho-sarcoma of the thymus gland, and this pathological finding has been exactly confirmed in Hun's case. Hun also refers to a case of Link's (27), in which there was present lymphoid infiltration of the muscles, with a persistent thymus, and in Oppenheim's case (34) there was a sarcoma of the anterior mediastinum. In Goldflam's case (17) there was a lympho-sarcoma of the whole upper lobe of one lung, with lymphoid infiltration of the muscles, which had also been found in a piece of the deltoid muscle excised twelve months before death. Hun's view of the paresis in myasthenia is that it is due to some alteration in the end plate in the muscle fibre, due to a chemical change in the lymph bathing the muscle fibre, and dependent in some way on the lymphoid infiltration of the muscle. Now in diphtheritic neuritis the lesion is proved by microscopical examination to be a structural alteration or degeneration of the nerve-fibres from the periphery up to the intramedullary portion of the anterior roots, close to their origin from the anterior horn cells, as shown by Katz and Luce, Bruns, Batten (1), and others. Occasionally, however, nerve-cell changes have been found, as by Bolton (4), in the

nucleus ambiguus in a case dying from heart failure, by Preisz (35), and by Michell Clarke (31), who showed gross changes by Nissl's method in the anterior horn-cells and nuclei after diphtheria. It is, then, not difficult to picture to oneself cases of increasingly severe lesion of the nerve-fibres and anterior horn cells and bulbar nuclei, the slighter forms of which, in which the lesion is no doubt limited to the nerve-fibres entirely, recover entirely, as in the ordinary non-fatal form of post-diphtheritic neuritis. A more severe lesion would be associated with more or less gross changes in the anterior horn cells or bulbar nuclei, which on the one hand might recover after many months or years, according to the severity of the cell damage, as in Case 2, or on the other hand might remain permanent, or might even become slowly progressive, as in Case 1. This is the lesion I consider to have occurred in my two cases, the greater cell destruction being in Case 1, perhaps irrecoverable, but certainly more severe than in Case 2, on account of the greater weakness, atrophy, and reaction of degeneration. Since, however, the second case recovered after the paralysis had persisted for six years, and, curiously enough, recovered after the birth of her first child, it is, perhaps, not impossible that Case 1 may also improve, as the symptoms have been present only four years. She married recently, in December, 1903, and it will be very interesting to observe the effect on the paralysis, should she become pregnant and give birth to a child. If I have an opportunity, I hope to publish the sequel of this case in BRAIN at a later date.

In conclusion, it seems clear that, very occasionally, permanent palsy may result following diphtheritic paralysis. That when such permanent paralysis remains, it is usually of muscles supplied by one or more of the bulbar nuclei. That a distinct type of bulbar paralysis may ensue, closely resembling in its distribution that which is familiar in myasthenia, or asthenic bulbar palsy, but which is to be distinguished from the latter by the non-variability of the symptoms, the absence of the myasthenic reaction, the absence of ptosis, or weakness of the jaw muscles or of the neck or limbs. It is further distinguished by the absence

of the attacks of dyspnœa so characteristic of many cases of myasthenia, and by the presence of muscular atrophy and the reaction of degeneration.

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THE DEGENERATIONS RESULTING FROM LESIONS OF POSTERIOR NERVE ROOTS AND FROM TRANSVERSE LESIONS OF THE SPINAL CORD IN MAN. A STUDY OF TWENTY CASES.

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HAVING held successively the post of pathologist to the National Hospital we have had the opportunity of examining (1) two cases in which there were isolated lesions of the posterior roots in the cervical or lumbo-sacral region, and (2) twelve cases of transverse lesion of the spinal cord, at various levels. In all of these cases the method of Marchi was applicable.

In connection with the descending systems of the posterior columns we have also made use of several cases of transverse lesion of the spinal cord, which we have examined by the Weigert-Pal method.

For the Marchi method we have invariably used Busch's sodium-iodate process. Inasmuch as the literature of the subject is very extensive, we have deemed it convenient to refer only to the more recent investigations concerning such anatomical and physiological points as have been but lately brought to light, or as are still debatable, and to which our observations may add some further information. A bibliography of the more recent literature upon these subjects is appended.

The subject matter of this paper is arranged as follows :—

The posterior roots.—(1) The descending intraspinal prolongations and their relations to the coma tract, to the septo-marginal system, and to other posterior descending systems. (2) The ascending intramedullary prolongations ; Lissauer's zone ; the question of direct and crossed fibres passing above the posterior column nuclei to the cerebellum.

Degenerations resulting from transverse lesions.—(1) Descending—The mode of termination of the pyramidal tracts. The descending degeneration in the lateral limiting zone. The posterior descending system. The comma tract ; septo-marginal system ; sulco-commissural tract ; septo-ventral fibres ; the ending of the sacral triangle. (2) Ascending—The ascending system of the dorsal longitudinal bundle. The ascending system of the posterior columns.

The direct cerebellar tract — Fibres arising in the cervical region. Fibres joining this tract in the medulla. The ventral and dorsal medullary plexuses of Horsley and Thiele. The connection with Gowers' tract. Termination in the cerebellum.

The spino-vestibular system of Horsley and Thiele.

The ventro-lateral degeneration in the spinal cord.

Gowers' tract.—Direct fibres to the flocculus ; connections with the nucleus lateralis inferior ; connection with the nucleus cerebello-acusticus, Bechterew's nucleus and Deiters' nucleus, by means of fibres arising in the anterior part of the velum. Termination in the cerebellum.

The prolongations of Gowers' tract above the pons.—The dorsal and lateral spino-tectal systems. The question of fibres passing to the substantia nigra. The spino-thalamic system. Absence of fibres going to the nucleus lenticularis.

The ventro-lateral restiformal fibres of Tschermak.

The dorso-olivary fibres of von Solder.

Certain possibilities concerning the degeneration of different parts of the axon, and especially of the collaterals, as revealed by the Marchi method.

The Degenerations Resulting from Lesions of Posterior Roots.

Case 1.—*Lesion of third lumbar posterior root on right side—examination by Marchi (Busch) method—intramedullary fibres traced upwards to the nucleus gracilis and downwards to the coccygeal segment of the cord.*

The patient was a woman who died in the National Hospital from carcinoma of the vertebral column secondary to mammary scirrhus, the primary disease having been removed three years previously. At the autopsy the lower part of the vertebral column was found extensively infiltrated by the growth, and here and there a nodule had invaded the lumen of the vertebral canal at the level of the cauda equina.

One of these nodules had caught and compressed the third lumbar posterior root of the right side just before its entry into the intervertebral foramen. The cord and medulla oblongata were examined by the Busch and Weigert-Pal methods. The latter showed no change from the normal. Sections from the third lumbar segment stained by Busch's fluid showed the right posterior root completely degenerated, and the entry root zone full of degenerated fibres. A very few degenerated fibres could be traced in the direction of the anterior horn, and one or two towards Clarke's column, which was only sparsely represented at this level.

The remainder of the section contained no degeneration.

The ascending degeneration followed the usual course and requires very little comment: fibres running into the gray matter were seen for a segment or two, but then became scarce, and there was no evidence anywhere of fibres crossing to the contralateral side of the cord. The upward termination was entirely in the nucleus gracilis, and no degenerated fibres could be traced among the arcuate bundles, internal or external. It should be noted that the region of Lissauer's tract presented very few degenerated fibres, even at a level immediately above the entry of the third lumbar root.

The degenerated descending collaterals were seen to be numerous and in the upper part of the fourth lumbar segment were still confined to the entry root zone. A little lower they began to spread out in a streak running antero-posteriorly, and this streak gradually approached the mid-line until, at the level of the first sacral segment, the fibres could be seen lining almost continuously the posterior median septum. In their course to this position they appeared to be divided into three principal groups, an anterior, a median and a posterior, but the separation between them was by no means distinct. A continual leakage of fibres took place from the anterior group into the gray matter at the base of the posterior horn, so that the number of degenerated fibres somewhat rapidly diminished. In a section from the fourth lumbar segment about five hundred dots of blackened myelin could be counted in the posterior column, whereas in a section from the fourth sacral segment only about eighty-five could still be distinguished.

Below the first sacral segment the degenerated fibres gradually approached the posterior extremity of the median septum and eventually became bunched in the septo-marginal angle forming one half of the well-known sacral triangle. In this region they were traced in rapidly diminishing numbers as far as the coccygeal segment, and their termination at different levels in the gray matter of the sacral cord was suggested by the appearance of a few scattered dots between the periphery and the posterior commissure. No evidence was forthcoming that these descending fibres ever crossed to the opposite side of the cord.

Case 2.—*A lesion of the extra-thecal roots of the cauda equina—examination of the cord by the Marchi* (*Busch*) *method—ascending degeneration as high as the gracilis nucleus—few descending fibres — well-marked degeneration of the posterior root fibres going to the anterior horns.*

The patient was a girl, 14 years of age, who, in January, 1900, began to suffer from sharp pains in the lower extremities. At the end of May she exhibited atrophic palsy of the legs and some sensory loss in the

lower lumbar and upper sacral areas. The sensory loss spread until the end of June, when the girl died of sarcoma of the pelvis.

At the autopsy the lower lumbar vertebræ and the entire sacrum were infiltrated by the growth, which involved the extrathecal portion of the roots of the cauda equina, but did not penetrate within the theca. All the posterior roots as high as and including the second lumbar on both sides, with the exception of the third lumbar on the right, showed well-marked degeneration by Busch's method. At the level of the fifth sacral segment both entering roots were degenerated, and the blackened fibres were seen occupying the greater part of the posterior column. It was noticeable, however, that only a few fibres were degenerated in the region known as the sacral triangle, and that the cornu-commissural zone was less affected than the middle root zone. All the entering roots as high as the fourth lumbar were markedly degenerated on both sides, and all contributed their share to the degeneration of the posterior column, but the distribution of the fibres remained very much the same as was seen in the fifth sacral segment.

The sacral triangle, which gradually merged into the oval area, presented a marked contrast, by reason of its pallor, to the surrounding parts, although it always contained a certain number of black dots. The ventral field, although not so pale, was less degenerated than the middle and posterior parts of the column. About the upper sacral region another comparatively undegenerated area appeared in the form of a small triangle, lying immediately internal to the entering root on the posterior periphery of the cord.

At the level of the third lumbar segment the posterior root on the right side was seen to contain but little degeneration, and, as a consequence, the external root zone was much paler than on the left side. In the second lumbar segment this pale area had been displaced mesially by the degenerated root of that segment, and lay in the form of a crescent about a third of the distance between the posterior horn and the posterior median septum. As high as this level numerous degenerated fibres could be seen coursing

far into the anterior horns and breaking up in the neighbourhood of the ganglion cells of that part.

The oval area in the second lumbar segment was less distinct than in the segment immediately below, and disappeared altogether a little higher; it could not be traced spreading out on to the surface of the cord.

Lissauer's tract contained a few very fine fibres, which disappeared rapidly at higher levels.

Throughout the thoracic region the degenerated fibres on both sides were separated from the posterior horns by a considerable and gradually increasing undegenerated field. They occupied an area extending from the periphery to the posterior commissure, the anterior portion expanding at first in the commissural region, but later receding towards the median septum until, at the level of the sixth cervical segment, the most anterior fibres did not reach the gray matter, and the whole degenerated field assumed the shape of a triangular wedge, with its base lying on the surface. Above this the degeneration was limited to Goll's column, and ended in the region of the nucleus gracilis. A very few fibres could be traced laterally above this in the direction of the corpus restiforme, but they broke up near its internal aspect. No degeneration could be traced into the internal arcuate fibres.

The Descending Intramedullary Prolongations.

The existence of descending posterior root fibres has been shown in human embryos by histological methods and in the adult by means of the degenerations which follow extramedullary root lesions. Their presence in animals (dogs, guinea pigs, &c.) has been proved experimentally.

The questions which remain to be answered are: (*a*) Do they exist in large or small numbers? (*b*) What is their length? (*c*) What is their course? (*d*) What is their relation to descending fibres of endogenous origin? and (*e*) What is their mode of termination?

Cases of a single extramedullary root lesion, which can be examined by the Marchi method, and which are not

complicated by other intramedullary degenerations, are of some rarity, so that the one reported above affords data of considerable importance.

In the first place it proves that the third lumbar posterior root contains descending intraspinal prolongations in large numbers. In the second place, it shows that some of these fibres are short, some of medium length, and some long, the shortest terminating in the gray matter of the fourth lumbar segment, and the longest traversing seven or eight segments of the lumbo-sacral cord, to end in the lowest parts of the gray matter. Both in their course and in their length the fibres of the third lumbar root correspond closely with those of the fourth lumbar root, the degeneration of which Wallenberg has described in one of his cases. Moreover, that author has shown that still lower roots send fibres caudalwards, and that of all those roots—sacral, lumbar, and perhaps lower thoracic—which send fibres to the lower sacral region of the cord, the descending fibres of higher origin tend to take a position mesial to those of lower origin. They provide, in fact, an analogy in their course to that of the ascending root fibres; those of the latter with the lower origin assuming a position mesial to those of higher origin in their passage upwards through the columns of Burdach and Goll.

Comparing our case with those described by Nageotte, Homen, Dejerine and Thomas, Wallenberg, Zappert, &c., it seems to be very clearly shown that descending fibres of a posterior root in the cervical and in the upper five-sixths of the thoracic region follow a somewhat different course to that of similar fibres with their origin in the lumbo-sacral roots.

In both cases immediately below the entering root the fibres are bunched up in the external root zone; in both cases, too, they quickly spread out into a somewhat narrow band, with its anterior limit in the region of the cornu-commissural angle, and its posterior limit on or near the dorsal periphary of the cord. This band is rapidly displaced mesially by other entering roots. In the case of the cervical and thoracic fibres they assume a position between the

columns of Burdach and Goll; in the case of the lumbo-sacral fibres they quickly reach and lie alongside the posterior median septum. The cervical and thoracic fibres can be traced through several segments — the cervical through more than the thoracic—in diminishing numbers. The lumbar and sacral fibres enter into the sacral triangle, or dorso-medial bundle, and are thence traceable into the gray matter of the lowest part of the cord.

The difference in the path taken by the cervico-thoracic and the lumbo-sacral exogenous descending fibres is more apparent than real, and can be readily explained on anatomical grounds. The column of Goll becomes a distinct formation in the lower part of the thoracic region, and is, no doubt, responsible for checking the mesial course of fibres of higher origin. In the lumbo-sacral cord, on the other hand, there is no definite postero-internal column, and the descending fibres are free to reach the median line without having to pierce a compact bundle of ascending fibres enclosed in a septal sheath.

Although at first sight the descending fibres of the thoracic posterior roots would appear, from the smaller number of segments they traverse, to be of shorter length than those of the cervical, lumbar and sacral roots, the difference is probably not one of actual length, but due to the fact that the segments of the former region of the cord are of greater length than those of the latter.

The descending fibres of all posterior roots rapidly diminish in number as they pass caudalwards, and this diminution is undoubtedly due to the leakage of many of them into the gray matter in the cornu-commissural region, some of them passing to Clarke's column and some into the posterior gray matter of the same side.

Our second case is of much less importance in so far as it throws any light on the course of exogenous descending fibres. It resembles in origin others which have been published, notably those of Darkschewitsch, Dejerine and Spiller, Marinesco and Souques, Wallenberg and Goldstein, all of which were examined by the Marchi method. A comparison of the results obtained brings out two im-

portant facts. In the first place it affords ample evidence of the presence of descending exogenous fibres in the sacral triangle and in the region of the oval area. In the second place it emphasises a point to which we shall have occasion to refer again, namely, the unreliability of the Marchi method for the staining of degenerated collaterals. In reference to the latter point it is worthy of note that Marinesco and Souques consider that the collaterals of the posterior roots degenerate sooner than the main stems, and that consequently in late Marchi specimens the former cannot be traced.

Reviewing our results and comparing them with those of other observers we may summarise as follows :—

(1) In the cervical and thoracic regions of the cord the descending exogenous fibres occupy a somewhat curved narrow area, extending anteriorly to the cornu-commissural angle and posteriorly close to the dorsal periphery of the cord in the inner part of Burdach's column. They must, therefore, take a very considerable part in the formation of the so-called comma tract, and it remains to be proved whether or no the latter contains fibres of endogenous origin as well. (2) In the lumbo-sacral region the descending exogenous fibres rapidly reach the neighbourhood of the posterior median septum, and are present therefore in large numbers in the oval area of Flechsig and in the sacral triangle of Gombault and Philippe. (3) There is no evidence to show whether the peripheral bandalette of Hoche contains endogenous or exogenous fibres or whether it contains both,[1] but the consensus of opinion appears to be in favour of its endogenous origin. Its undoubted continuity with the oval area and sacral triangle, as shown by Bruce and Muir, suggests that those areas contain endogenous and exogenous fibres, supposing the view that the bandalette arises from cells within the cord is correct. (4) The ventral field of the posterior columns contains exogenous as well as endogenous fibres, but the former are

[1] We are unable to accept as conclusive the results obtained from the study of cases of tabes dorsalis by the Weigert-Pal method, since the degeneration in that disease does not follow exactly root distributions, and in many cases includes fibres of undoubted endogenous origin.

less numerous than the latter, and are more limited to its median part. (5) In the lumbo-sacral region a small triangular area, lying just internal to the entering posterior root on the dorsal surface of the cord, is composed mainly of descending endogenous fibres mixed with very few exogenous collaterals.

The Ascending Intramedullary Prolongations.

We do not propose to describe in detail the course of the ascending exogenous fibres, as our findings have not differed from those of previous observers. We may state, however, that we are unable to trace the very fine fibres of Lissauer's zone for more than a segment or two above a degenerated posterior root, and that even in that short course they become very few and far between. In neither of our cases could we trace any fibres passing above the posterior column nuclei to the cerebellum.

Degenerations Resulting from Transverse Lesions. (1) Descending Degenerations.

The Termination of the Pyramidal Tracts in the Lower Spinal Cord.

The termination of certain fibres of this tract in Clarke's nucleus was very well shown in several of our cases of transverse lesion. That the degenerate fibres passing from the lateral column to Clarke's nucleus were pyramidal fibres was proved by their similarity in position and level to the degenerate fibres in a case of recent hemiplegia which we have examined.

It is so rare to find the terminations of this tract well stained with the Marchi method that we think a detailed description is not out of place in this paper.

We may add that of sixteen cases of hemiplegia which we have examined by the Marchi method, the terminations of the pyramidal tract were only demonstrable in two cases. The same was true of the majority of the cases of transverse lesions.

Few fibres could be traced out of the pyramidal tracts above the tenth dorsal segment. In the tenth, eleventh and especially in the twelfth dorsal segments, and in the first and second lumbar segments, coarse fibres passed transversely inwards to the front of Clarke's column. So directly transverse was their course that in sections 100 μ thick they could be followed from the pyramidal tract to Clarke's nucleus in the same section. Arrived at the front of Clarke's nucleus these fibres turn abruptly downwards and run for some few segments in contact with the nucleus, fine and coarse collaterals continually entering and breaking up among the cells of the nucleus.

Very few fibres passed inwards from the pyramidal tract below the second lumbar segment, but the fibres coursing down along the front of Clarke's nucleus were visible in the posterior gray matter at the first sacral segment.

No fibres could be traced to any part of the ventral horns.

We would call attention to that peculiarity in the degeneration of the distal portions of long neurones which renders them apt not to stain by the Marchi method, as the explanation of the difficulty which has been general in determining the termination of the pyramidal tract in the spinal cord.

The Degenerations in the Lateral Limiting Layer.

As a result of investigation by the myelination method and from the study of a case of amyotrophic lateral sclerosis, it has been suggested by Flechsig and by Bruce that the dorsal division of the lateral limiting layer represents ascending fibres of short intraspinal course, while the ventral division represents descending fibres of short intraspinal course.

We carefully sought in sections immediately above and below the lesions in all our cases for evidence of preponderant degeneration in the dorsal division above the lesion, and preponderant descending degeneration below the lesion, in the ventral division of the lateral limiting layer. We found only a scattered ascending and descending degeneration equally distributed over both divisions.

Descending Degeneration in the Posterior Columns.

The results obtained in all the cases examined by the Marchi method were constant, and may be briefly summarised as follows:—

Immediately below the region of the "traumatic degeneration" the degenerate fibres in the posterior columns could be divided into three classes.

(1) A large band of mixed coarse and fine fibres in the postero-external column, reaching from the gray matter at the junction of the posterior horn and posterior commissure to the surface of the posterior column, separated except at its ventral extremity from the posterior horn by a considerable mass of non-degenerate white matter. This degenerate strip extended downwards in the same position in the postero-external column for a distance which varied directly with the position of the lesion in the spinal cord; for instance, with the lower limit of the lesions in the eighth cervical, fifth and tenth dorsal segments, the degenerate band extended to the seventh, ninth, and eleventh dorsal segments respectively. The band then separates into (1) a ventral portion, lying against the dorsal horn (sulco-commissural bundle of Dufour), the majority of the fibres of which gradually pass into the gray matter at the junction of the dorsal horn and dorsal commissure. This tract finally disappears at the fifth lumbar segment. Some of the coarse fibres of this bundle pass mesially to reach the septum, and then keeping along the septum go dorsalwards to join the degeneration of the oval area. (2) A dorsal portion, many coarse fibres of which rapidly gain the dorsal surface of the posterior column, where they subsequently spread out to form the dorsal crescent or peripheral bandalette of Hoche in the twelfth dorsal segment, and passing on to the edges of the septum at the second lumbar segment form the oval area of Flechsig, which exists throughout the third, fourth, and fifth lumbar segments. These fibres passing again dorsally in the second sacral segment form the sacral triangle of Gombault and Philippe at the third sacral segment, and from this to the end of the conus medullaris

they stream into the posterior gray matter of the same side to break up among a mass of nerve-cells in a position similar to that of the "sacral nucleus." This group of fibres is that to which Bruce and Muir have applied the term "septo-marginal tract." In addition to these fibres, we desire to draw attention to certain fine fibres of the dorsal division of this system which do not join the posterior crescent, but which remain throughout the greater part of their course in Burdach's column close to the dorsal surface.

(2) Hoche's septal fibres. Immediately below the lesion we have in all the cases found a conspicuous row of somewhat large fibres lying almost in single file along either side of the ventral one-third of the septum, but not reaching to the commissure. These fibres pass along the septum into the gray matter in the region of the commissures. They do not extend for more than two segments below the lower limit of the lesion.

(3) Scattered degeneration of fine fibres throughout the posterior columns, situated chiefly in Burdach's column and in the external root zone. They disappear for the most three or four segments below the lesion.

We have now to draw attention to certain details of our observation.

The Descending Strip of the Postero-External Column.

It has been already stated that the higher the level of the lesion in the spinal cord the farther does this strip of degeneration descend as such before dividing into its dorsal and ventral groups. It extends downwards for seven segments below a cervical lesion, three segments below a mid-dorsal lesion and only one segment below an eleventh dorsal lesion. Further, in a lesion of the twelfth dorsal we found that the coarse fibres of the dorsal and ventral divisions were already separate immediately below the region of "traumatic" degeneration. While we believe that we are the first to state this fact definitely, yet the publications and figures of Hoche, Nageotte and all the

more recent observers, if taken collectively, bear out our conclusion.

The descending strip immediately below the lesion extends from the gray matter at the junction of dorsal horn and dorsal commissure to the dorsal surface of the dorsal column. It is limited externally by the external root zone, and is composed of very coarse fibres, medium-sized fibres and fine fibres. The very coarse fibres are situated mainly on the dorsal one third of the strip, but a few are found at its ventral limit. As the tract descends it tends to assume somewhat of an hour-glass shape, with a larger dorsal expansion reaching to the dorsal periphery in which the majority of the larger fibres are collected, a much smaller ventral extremity lying against the gray horn and a connecting portion composed mainly of fine fibres. From this central portion the fine fibres are continually running ventralwards to enter the gray matter in the cornu-commissural region, till at the eleventh dorsal segment they have all disappeared, leaving the ventral and dorsal group separate.

The ventral group (sulco-commissural bundle of Dufour) retains its position against the gray horn and, inclining towards the septum as it descends, disappears constantly in the fifth lumbar segment. Fibres are continually passing from it to break up in the contiguous gray matter. Some of the coarse fibres which it contains, however, pass to the septum and, running dorsalwards, join the oval area in the upper part of the third lumbar segment. We wish to call attention to this fact especially since in previous descriptions the oval area has been said to be derived entirely from fibres reaching the septum by the dorsal periphery. We are indebted to the kindness of Dr. Bruce, of Edinburgh, for the confirmation of our results, for he informs us that he has also found similar ventral fibres passing to the oval area.

All the coarse fibres of the dorsal division of the descending degeneration in Burdach's column pass on to the dorsal periphery of the cord in the lower part of the eleventh and in the twelfth dorsal segments to form the

posterior crescent, or peripheral bandalette of Hoche. Most of the fine fibres disappear, but we would draw attention to the fact that some fine fibres remain in the dorso-external region of the posterior columns, and can be traced in lesions from the mid-dorsal region downwards as far as the conus, where they course ventrally through the thickness of the posterior column to enter the gray matter. The subsequent course of the septomarginal system, the passage of the posterior crescent to the septum, the formation of the oval area and its connection with the sacral triangle, the fibres of which end in the dorsal gray matter of the same side, corresponded in our cases with the descriptions given by other recent observers. Whatever be the level of the transverse lesion, the following degenerate tracts assume their characteristic position at the following levels:—

Posterior crescent (bandalette)) Sulco-commissure bundle)	D. 12.
Reaches septum	L. 1, L. 2.
Oval area...	L. 3, 4, 5.
Sacral triangle	S. 3.
Enters gray matter	S. 5, Co.

In connection with the termination of the sacral triangle we may again draw attention to a fact we have already mentioned more than once in this paper, that except degeneration be very recent the distal portions of degenerate long neurones may not be revealed by the Marchi method. Whereas in many of our cases the fibres of the sacral triangle entering the gray matter were well shown even to their terminal ramifications, in other cases no sign of such entering fibres could be found, notwithstanding that the fibres of this triangle and the rest of the descending posterior degenerate fibres were well stained. For example, two cases of transverse myelitis at the level of the ninth dorsal were examined. In one death had occurred five weeks after the onset, and the ending of the sacral triangle was perfectly stained. In the other death had occurred eight weeks after the onset and the ending of the triangle was not stained.

The Anatomical Limitation of the Oval Area and the Sacral Triangle by Fibrous Septa.

A most striking feature of many of our preparations has been the separation of these tracts at certain levels by fibrous septa.

As the fibres of the posterior crescentic degeneration pass ventrally at the level of the second lumbar to form the oval area, they split the posterior septum into two layers, between which all the fibres of the oval area are contained. When from the oval area the fibres incline backwards to form the sacral triangle, these two layers are carried backwards on either side of the dorsal piece of the posterior septum, so that at the level of the second sacral segment each half of the sacral triangle is separated off. These septa become less distinct at the third sacral level when the sacral triangle becomes fully formed.

The Scattered Posterior Degeneration below the Lesion.

This cannot be accounted for as "traumatic" degeneration. Some of the degeneration in the external root zone doubtless corresponds with descending collaterals of the posterior roots. Certainly much of it is derived from fibres passing away from the main descending degenerate tract in the postero-external column.

Hoche's Ventral Septal Fibres.

In all the cases we have found these fibres lying in single file along the ventral one-third of the septum, not reaching the gray matter and not extending more than two segments clear of the lesion. They are moderately large fibres and their number seems to be variable. On the whole they seem to be more numerous in cases of cervical and upper dorsal lesions than in those lower down. But in one case of lesion at the tenth dorsal we found as many as in any of our cervical cases. In the cervical and upper dorsal cases we could trace them for two segments, lower down for only one segment, while in the lowest lesion we examined (twelfth dorsal) they were breaking up in the

upper part of the segment below the lesion. Their mode of termination was clear in several of our cases. They pass ventrally along the septum towards the gray matter, breaking up and spreading out as they do so, so that just before their disappearance they are seen as a multitude of fine points in the septo-commissural angle.

(2) Ascending Degenerations.

Ascending System of the Dorsal Longitudinal Bundle.

In all the cases of transverse lesion of the spinal cord degenerate fibres were found throughout the length of the dorsal longitudinal bundle above the lesion. The majority of those fibres were fine fibres and few coarse fibres were found above the level of the sixth nucleus. In the cases of low dorsal lesions only a few fibres were found. Where mid-dorsal lesions existed the number was distinctly greater, while in the cases of lesion of the cervical enlargement many more degenerate fibres were present. It would appear, therefore, that these fibres constitute a true ascending system arising in each segment of the spinal cord. The small proportion which the degenerate fibres of the dorsal longitudinal bundle bore to the non-degenerate fibres, even in lesions of the cervical enlargement, bears out the generally-accepted view that the majority of the fibres of this system are descending fibres and, in comparing our sections from this series of cases with a series showing descending degeneration in the dorsal longitudinal bundle as a result of experimental and pathological lesions of the brain stem, published in Brain, 1901, the paucity of the degeneration in the former contrasted most strikingly with the richness of the degeneration in the latter. The degenerate ascending fibres of the dorsal longitudinal bundle appear to take their origin in the ventral horn of the same side. A few segments above the lesion they lie in part mesial and in part ventral to the ventral horn. Traced upwards the majority incline outwards to enter the anterior extremity of the ventro-lateral area of degeneration, and are situated near the surface in the mid-cervical region.

In the first cervical segment they again sink towards the mesial aspect of the ventral horn and with the other degenerate fibres, which pass mesial to the inferior olive, form a characteristic band, arc-shaped in transverse section, stretching from the ventro-lateral region towards the mesial aspect of the ventral horn. When the pyramid commences these fibres lie between the pyramid and the remains of the ventral horn, being here joined by fibres of the same system which have retained a position close to the lips of the ventral fissure throughout their course in the spinal cord. Passing mesial to the olive among the outer fibres of the fillet, the dorsal longitudinal bundle fibres separate from the degenerate dorso-olivary fibres which accompany them and ascend rapidly to the dorsal white matter, on either side of the middle line, where they are so arranged that the fibres from the lower parts of the cord lay dorsal and mesial to those from the higher segments of the spinal cord, as judged from a comparison of the areas occupied by the degenerate fibres in lesions of the lower dorsal, mid-dorsal and lower cervical cord respectively.

We wish to draw especial attention to the large number of degenerate fibres, which have been present in all our cases, lying immediately external to the pyramid directly it formed and occupying the position of the dorsal longitudinal bundle. Most of these fibres do not belong to the system of the dorsal longitudinal bundle, but pass from the ventro-lateral degeneration of the cord mesial to the olive, some entering the inferior olive, the majority passing outwards into the dorso-olivary layer to end in the restiform body (ventro-lateral restiformal system of Tschermak) and a few passing to the mesial fillet (fibres of von Solder). Above the limit of the hypoglossal nucleus the degeneration in the dorsal longitudinal bundle becomes much less marked, and many of the coarse fibres disappear. The fibres were not traced into the hypoglossal nuclei, but were easily followed to the nucleus centralis inferior, thus corresponding closely with the *descending* ponto-spinal fibres which take their origin in these nuclei. Above this level the degeneration could be

traced into the sixth nuclei, where most of the coarse fibres disappeared; a few fibres could be followed to the fourth and third nuclei and to the nucleus of Darkschewitch. None were traced across the posterior commissure.

Ascending Degenerations in the Posterior Columns.

Both in our cases of dorsal root lesions and of transverse lesions our results coincided with the classic descriptions so far as the spinal cord and posterior nuclei are concerned.

We conclude with confidence, however, that there are no direct fibres passing to the cerebellum from the posterior columns by way of the posterior nuclei, neither as external nor as internal arcuate fibres, for we have failed to find them in any of the cases. We have, in several cases, found fibres passing to the gray matter mesial to the restiform body from these nuclei, and these could be followed some distance upwards in the descending vestibular root, thus confirming the observations of Horsley and Thiele.

The Direct Cerebellar Tract.

The degeneration of Flechsig's tract in our cases corresponded in great part with the generally accepted anatomical description. It seems certain from a comparison of the bulk of the degeneration in this tract at different levels, that each segment of the spinal cord contributes fibres to the system. Though many writers have pointed out this fact, the relative contribution made by the various regions of the cord has not been made clear. We judge from our preparations—(1) that a majority of the fibres have their origin below the twelfth dorsal segment; (2) that throughout the thoracic region there is decreasing contribution from each segment; (3) that there is again a large accession of fibres from the cervical enlargement. In the case of the lowest transverse lesion we examined (upper limit in tenth dorsal), there was a copious degeneration immediately above the lesion already occupying the position of the direct cerebellar tract, while for three segments above coarse fibres lying in the region between the posterior horn and the direct cerebellar tract streamed outwards into the latter tract. These presumably

were fibres from distal and proximal segments below the lesion respectively. When the degenerations in the medulla of this case were compared with those in which the upper limit of the lesion was in the fifth cervical, it seemed obvious that more than one-half of the fibres in this tract are of lumbo-sacral origin, and less than one-half of thoracic and cervical origin.

Comparing further the degenerations resulting from lesions with the upper limit at the ninth, seventh, sixth, fourth, and third dorsal segments, the bulk of the degeneration above each lesion further cephalwards became moderately greater, while a comparison of the degeneration of the case of the highest lesion we have examined (upper limit at the fifth cervical) with that of the third dorsal lesion showed that a very large number of fibres had been added to the tract from the lower part of the cervical enlargement.

When describing the degenerations in the lateral column above transverse lesions of the lower and mid-thoracic region, we refer later to the dorsal of two angular areas of degeneration projecting inwards from the more definite dorso- and ventro-lateral degenerations of the direct cerebellar tract and Gowers' tract as being probably composed of fibres passing to the direct cerebellar tract. If this suggestion prove correct these fibres are added to the ventral and mesial aspect of Flechsig's tract, and it would seem likely that the longest fibres of this tract are situated dorsally and externally, while the shorter fibres have a position which is ventral and mesial relatively to their length.

Our observations fully confirm the opinion of Horsley and Thiele that the fibres of the direct cerebellar tract can be distinguished from those of Gowers' tract and other systems in the ventro-lateral region by their greater size. The spino-vestibular fibres, however, seem to be coarse and to occupy the dorsal limit of Flechsig's tract. We wish to point out that deductions from the size of fibres are apt to be entirely fallacious if the degeneration is not quite recent, and if the paraffin method be employed.

At the lower part of the first cervical segment the direct cerebellar tract occupies an even strip along the surface of

the cord from the posterior horn to the attachment of the denticulate ligament. Above this the inner edge of the tract is thrown into several folds as the fibres of the tract pass backwards to occupy the angle between the Rolandic substance and the lateral surface of the cord.

At the level of the commencing decussation of the pyramid a tongue-like mass of coarse fibres is seen to extend dorsalwards from the direct cerebellar tract between the Rolandic substance and the surface. This tongue-shaped process seems to us to constitute an anatomical separation between the spino-vestibular fibres of Horsley and Thiele, of which it is composed, and the direct cerebellar tract.

It is important to point out that at this level the lateral ascending degeneration is divided up into more or less completely separated groups—the spino-vestibular group, the direct cerebellar tract, Gowers' tract, and the dorsal longitudinal bundle groups.

In the medulla the spino-vestibular fibres of the direct cerebellar tract are the earliest to pass off as external arcuate fibres and, running in the layer of gray matter covering the restiform body, pass over the dorsal surface of that structure and turn ventralwards to break up in the gray matter about the descending vestibular root.

In several cases a few coarse fibres of the direct cerebellar tract were seen passing ventral and then mesial to the spinal trigeminal root. They have been regarded by Horsley and Thiele as fibres of the tract taking an unusual course. It is possible, however, that some of them end in the gray matter mesial to the spinal trigeminal root.

In the medulla the direct cerebellar tract gives off numerous collaterals to the ventral collateral plexus and to the dorsal collateral plexus, to be subsequently described. It receives coarse fibres from the degeneration of the dorso-olivary layer. These fibres come from the ventro-lateral degeneration of the cord and pass up mesial to the inferior olive, with the fibres of the dorsal longitudinal bundle subsequently crossing by the dorso-olivary layer to the restiform body.

As the restiform body enters the cerebellum we were

able to trace coarse fibres to the peduncle of the flocculus in every case.

Arriving beneath the dentate nucleus the degeneration in the restiform body became divided into two, a smaller mesial bundle and an outer larger bundle. The mesial bundle turned almost vertically dorsalwards and passed mesial to the dentate nucleus giving off fibres to this nucleus, and ran partly ventrally, partly through the root nuclei, to terminate in the inferior vermis. Some fibres crossed both in the interfastigial commissure and in the inferior commissure.

The lateral bundle passed outside the dentate nucleus, giving off many fine collaterals to the fleece of that nucleus.

Just before the outer surface of the superior peduncle becomes free this bundle receives a considerable accession from Gowers' tract, which is here in close proximity to it. The fibres of the lateral bundle terminate chiefly in the superior vermis, many crossing the middle line in the anterior commissure, some in the posterior commissure. Degenerate fibres were traceable to all parts of the cerebellar cortex, especially to the lower part of the lateral lobe.

The Collateral Medullary Plexuses of the Direct Cerebellar Tract.

These plexuses were first figured by Hoche, and were described in detail by Horsley and Thiele. They were present in all of our cases of transverse lesion, and our results entirely confirm the opinion of Horsley and Thiele that they are composed of collaterals of the external arcuate fibres of the direct cerebellar tract, and that they have no connection with the degeneration in the posterior column nuclei, as has been suggested by Barker. The following description is that of Horsley and Thiele, to which we have added certain deductions from our observations :—

Ventral collateral plexus.—"Just above the uppermost level of the decussation of the pyramid strong collaterals, springing from the fibres of the direct cerebellar tract as they turn up towards the restiform body, pass inwards, first ventrally to the spinal trigeminal root, and between that

and the antero-lateral nucleus, breaking up and apparently terminating in connection with small nerve-cells lying just ventral to the spinal trigeminal root; the uppermost limit of the plexus is the upper border of the auditory striæ."

In many of our cases we have found fibres passing inwards from the mesial aspect of the direct cerebellar tract as low as the caudal end of the pyramidal decussation, which appear to break up in that part of the central gray matter corresponding with the nucleus accessorius. Perhaps these are the fibres which have been described by Kohnstamm as passing over from the direct cerebellar tract to the pyramid.

We wish to suggest that these fibres belong to the system of the ventral collateral plexus, the lower limit of the latter being the lowest limit of the pyramidal decussation.

From our specimens it seems likely that fibres of Gowers' tract enter into this ventral plexus from the funiculus proprius lateralis medullæ.

Dorsal collateral plexus.—"Commencing at the place of the upper limit of the pyramidal decussation, and limited above by the level of the highest auditory striæ, fine collateral fibres arise from the direct cerebellar tract and pass inwards, partly through the spinal trigeminal root, to end in a fine plexus just external to the gray nucleus enveloping the fasciculus solitarius."

This plexus is very well marked in our preparations, and in several cases we find fibres actually entering the fasciculus solitarius, which we could trace for some distance upwards; these subsequently re-entered the gray matter and disappeared.

In connection with these collateral plexuses we wish to point out that, unless the degeneration is very recent, these plexuses are apt not to be revealed by the Marchi method. Degenerate collaterals lose their property of reacting much sooner than do coarse fibres, and further, unless the degeneration is quite recent, alcohol, ether and clarifying reagents tend to dissolve out the black substance from the collaterals. This fact, first pointed out by Marinesco, has been fully

confirmed by our observations, and is most important since it adds greatly to the difficulty of correctly determining the ultimate destinations of degenerate fibres in pathological material.

As an example, we have found no sign of these plexuses in one case where the degeneration of the direct cerebellar tract was well marked and stable, the tissues being prepared by the celloidin method and mounted in balsam; yet portions from the same medulla cut with gum embedding and freezing, and examined immediately in glycerine, showed the plexuses very well.

The Spino-Vestibular Tract.

This system of fibres, to which attention was first drawn by Horsley and Thiele, was distinct in all our cases of transverse lesion of the spinal cord.

The fibres are coarse fibres which occupy the most dorsal part of the direct cerebellar tract. They appear to have their origin in the lumbo-sacral region, for we were unable to find a larger number of the fibres of this system degenerate in lesions at cervical levels than occurred in lesions of the lowest thoracic segments. Their position at the dorso-external limit of the direct cerebellar tract also suggests that they are all lumbo-sacral fibres.

These fibres are partly separated from the direct cerebellar tract in the upper part of the first cervical segment, where, as seen in transverse sections, they form a tongue-like projection extending dorsally from the direct cerebellar tract between the Rolandic substance and the surface of the cord.

They pass away from the cerebellar tract as the earliest of all the external arcuate fibres to be formed, and, curving round the restiform body in the thin layer of gray matter covering that structure, they sink almost perpendicularly, to break up in the gray matter mesial to the restiform body, which is associated with the descending auditory root. In this region, also, certain fibres end which pass outwards from the gracile and cuneate nuclei, and are derived from the degenerate ascending systems of the posterior columns.

The Ascending Degeneration in the Ventro-Lateral Region of the Spinal Cord.

Immediately above the area of traumatic degeneration in lesions of the thoracic cord the direct cerebellar tract was conspicuous as a narrow strip lying against the lateral periphery and reaching from the dorsal horn to the attachment of the denticulate ligament. Gowers' tract was also plainly visible, and we can fully confirm the observation of Risien Russell that it is usually separated from the surface of the lateral column by a layer of non-degenerate fibres.

We locate this tract as ventro-mesial to the ventral part of the direct cerebellar tract.

These two fibre systems can readily be distinguished by the large size of the fibres in the former tract.

A few segments above the lesion the mesial limit of the conspicuous ventro-lateral degeneration had the form of two hook-like processes, with their concavities directed dorso-mesially. The dorsal of these two processes was the larger, and contained the coarsest fibres; it was apparently limited behind by the lateral pyramidal tract. We suggest that it is composed of fibres passing to the direct cerebellar tract. It disappears about six segments above the lesion.

The ventral hook-like process was smaller and contained finer fibres than the dorsal process. It was generally visible as high as the second dorsal segment. We suggest that it is composed of fibres passing to Gowers' tract.

The scattered degeneration in the ventral column gradually diminished as it was traced upwards and tended to assume two positions: (1) in front of Gowers' tract and close to the surface, where it reached to the upper cervical region, extending almost as far round the ventral column as the mouth of the fissure; (2) along the lips of the ventral fissure. The fibres of this degeneration were mostly fine fibres, but some coarse fibres existed. The prolongations to the medulla and brain stem were the spino-tectal and spino-thalamic systems, the fibres of Tschernak, von Solder's fibres, and the ascending fibres of the dorsal longitudinal bundle.

Gowers' Tract and the Associated Systems.

In the spinal cord the fibres of these systems could be distinguished from those of the direct cerebellar tract by the greater size of the fibres of the latter. If the size of the spino-thalamic fibres in the mesencephalon was compared with that of those fibres of Gowers' tract which pass to the cerebellum, the latter were obviously of greater calibre than the former, but such difference did not enable us to distinguish these systems one from the other in the spinal cord nor in the medulla. Horsley and Thiele locate the spino-thalamic and spino-tectal systems as mesial to the other fibres of Gowers' tract in the funiculus lateralis proprius medullæ, and while it is true that the mesial fibres degenerate in that situation are of finer calibre than the others, we were unable to come to a definite conclusion upon this point. Certainly no separation could be made in the pons. For a few segments above a lesion in the mid and lower thoracic region the mesial outline of the ventro-lateral area of degeneration took the form of two angular projections, slightly hooked, with the concavity directed dorso-mesially. We have referred to the posterior of these as probably consisting of fibres passing to the direct cerebellar tract, and we suggest that the ventral projection which is composed of finer fibres is made up largely of fibres passing to Gowers' tract. From the comparison of the number of fibres degenerate in this tract as it traverses the medulla and pons, in cases of transverse lesions at various levels of the spinal cord, it seemed certain that fibres are added to the tract from each segment of the cord.

Especially would we point out that the number of spino-tectal and spino-thalamic fibres was considerably greater in lesions of the cervical enlargement than in lesions at lower levels.

While there is no doubt that many fibres occupying the position of Gowers' tract are internuncial fibres which enter the gray matter of the ventral horns at various levels, and that a gradual decrease of the bulk of the degeneration

Description of Plates.

Plate I.

From Case 1 in text. Fig. 1 shows the degenerated third lumbar posterior root entering the spinal cord. Fig. 2 is a photograph of the posterior columns of the fourth sacral segment, showing the degenerated descending root fibres lying alongside the posterior two-thirds of the posterior median septum.

Plates II. and III.

Drawings made by means of a projection apparatus in order to show the ascending and descending degenerations from a lesion of the third lumbar posterior root (Case 1.) The dorsal and cervical sections are drawn on a smaller scale than the lumbar and sacral.

Plates IV. and V.

Drawings showing the degeneration resulting from a lesion of cauda equina involving the posterior as high as the second lumbar on both sides, with the exception of the third lumbar on the right.

Plate VI.

Photograph of a section from the second lumbar segment of the same case.

Plate VII.

Fig. 1.—Transverse section of twelfth dorsal segment, showing degenerate fibres of the pyramidal tracts passing to the ventral aspect of Clarke's nucleus.

Fig. 2.—The same more highly magnified.

Plate VIII.

Transverse section of the pons and cerebellum, showing the degeneration in Gowers' tract, and in the direct cerebellar tract.

Plate IX.

Fig. 1.—A portion of the section shown in Plate VIII. more highly magnified, and showing the degenerate fibres passing to Bechterew's and Deiters' nuclei.

Fig. 2.—Transverse section at the level of the commencing pyramidal decussation.

Plate X.

Fig. 1.—Transverse section of the third dorsal segment, showing the two projections of the ventro-lateral ascending degeneration. (Lesion at eighth dorsal.)

Fig. 2.—The comma tract at the level of the tenth dorsal segment. (Lesion at eighth dorsal.) Weigert-Pal method.

Fig. 3.—The division of the comma tract in the twelfth dorsal segment. (Lesion at tenth dorsal.) Weigert-Pal method.

Fig. 4.—The fibres of the sacral triangle passing on to the gray matter of the fifth sacral segment.

Plates XI.—XVI.

Projection drawings of the ascending and descending degeneration, revealed by the Marchi method in a case of a total transverse lesion of the seventh dorsal segment.

PLATE I.

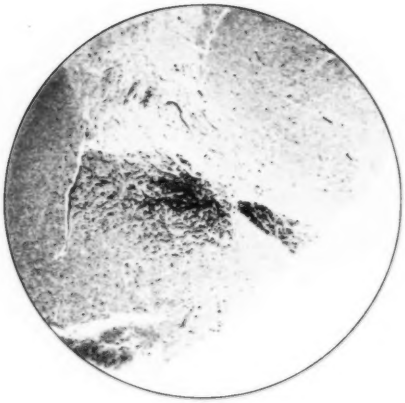


FIG. 1.

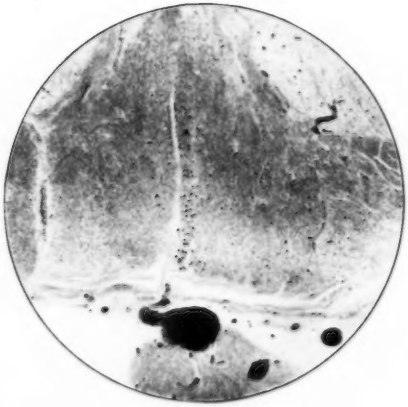


FIG. 2.

PLATE II

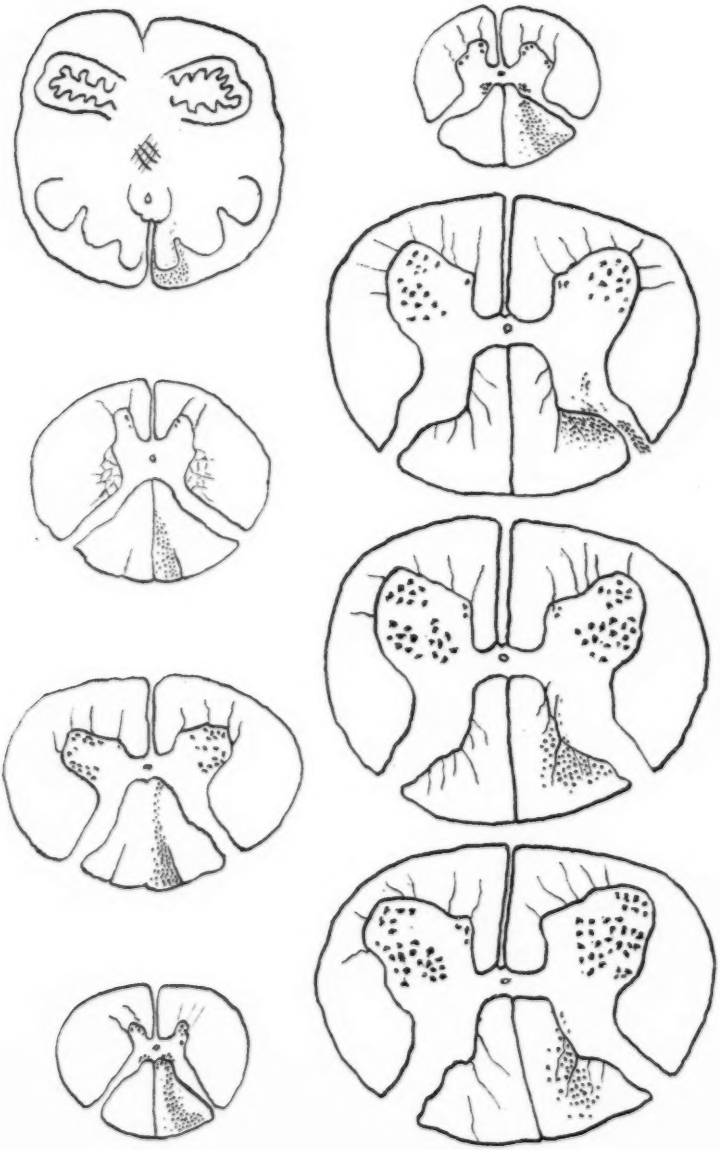


PLATE III

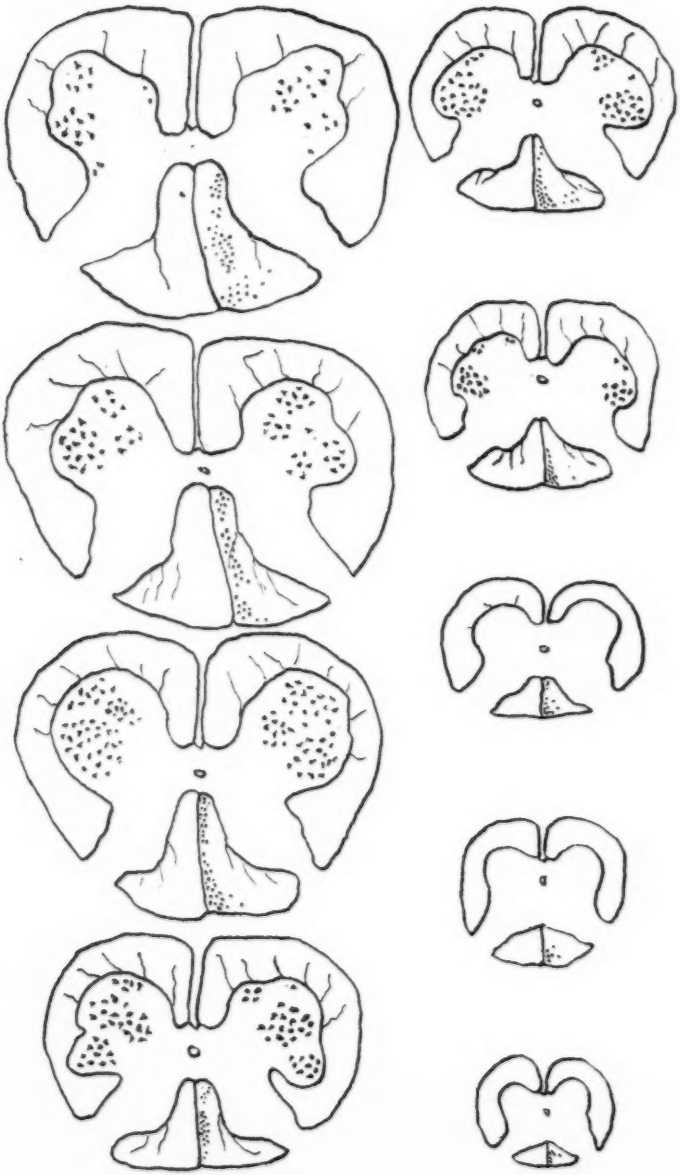
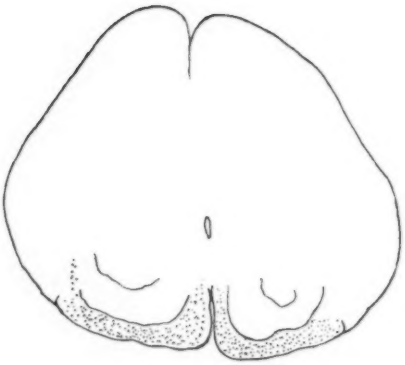
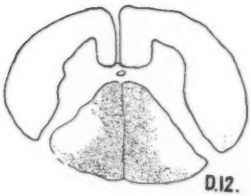
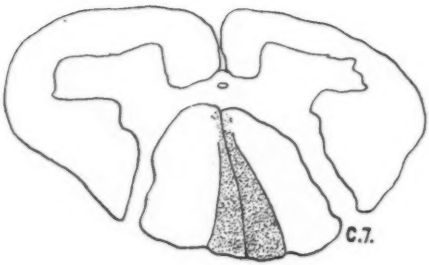
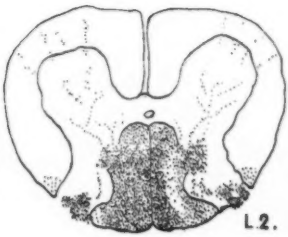


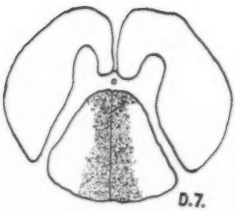
PLATE IV











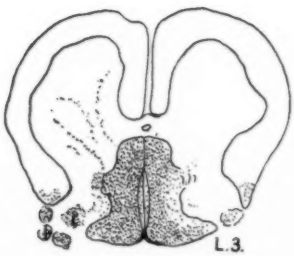
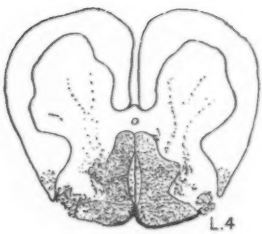
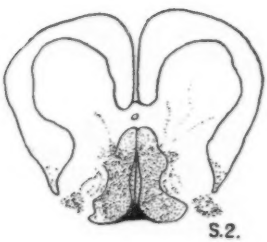
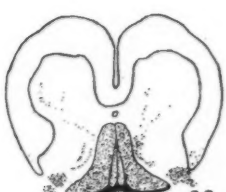
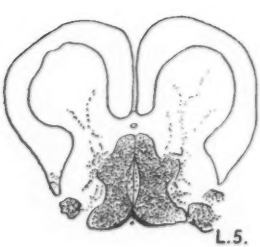


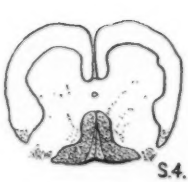
PLATE V

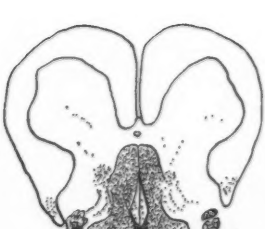












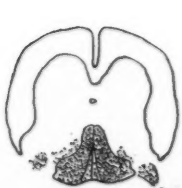


PLATE VI.

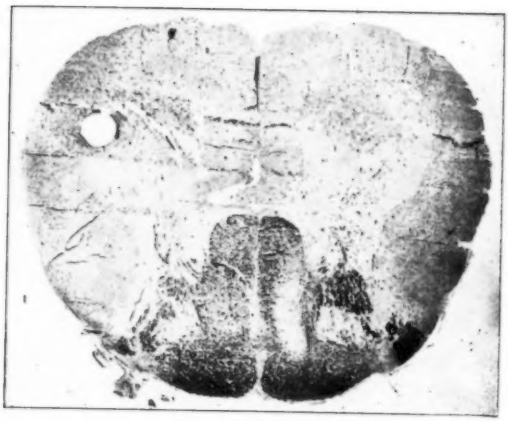


PLATE VII.

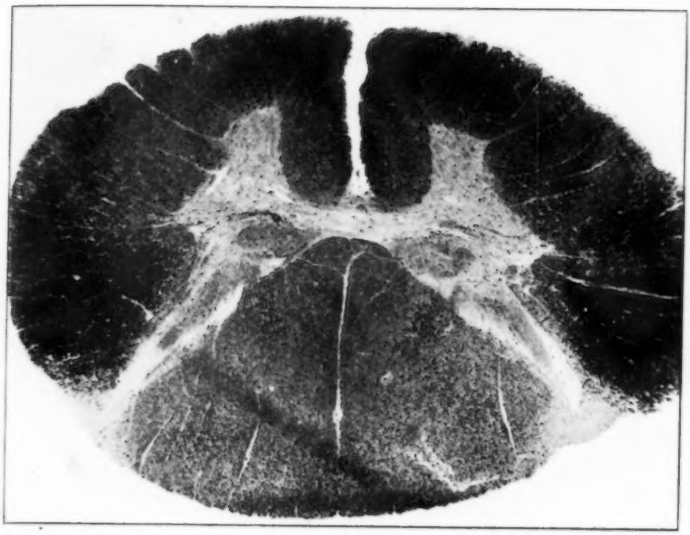


FIG. 1.

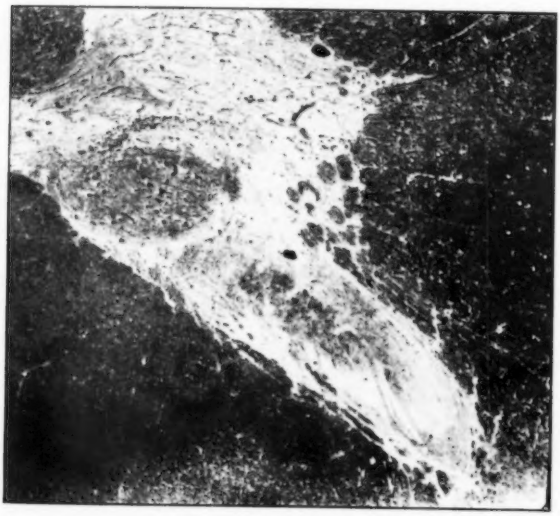


FIG. 2.

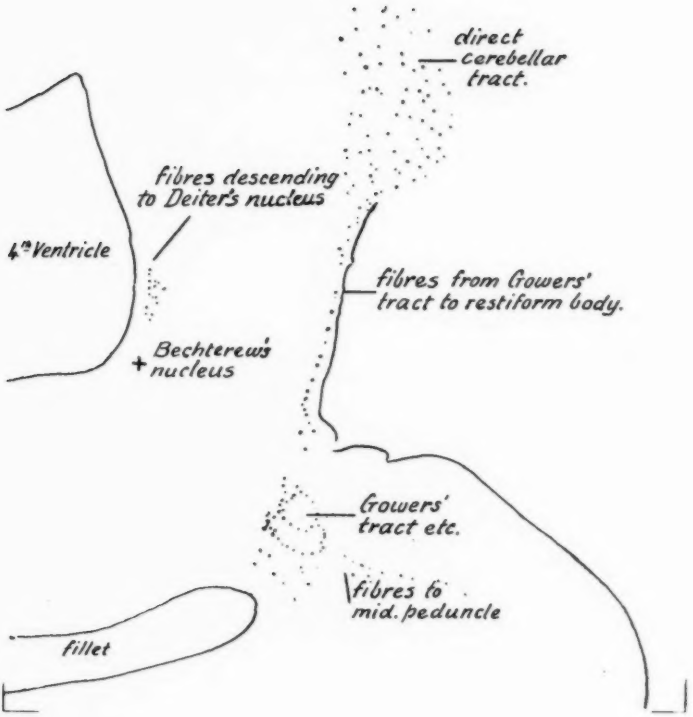

direct
cerebellar
tract.
fibres descending
to Deiter's nucleus
4th Ventricle
fibres from Gowers'
tract to restiform body.
Bechterew's
nucleus
Gowers'
tract etc.
fibres to
mid. peduncle
fillet

PLATE VIII.

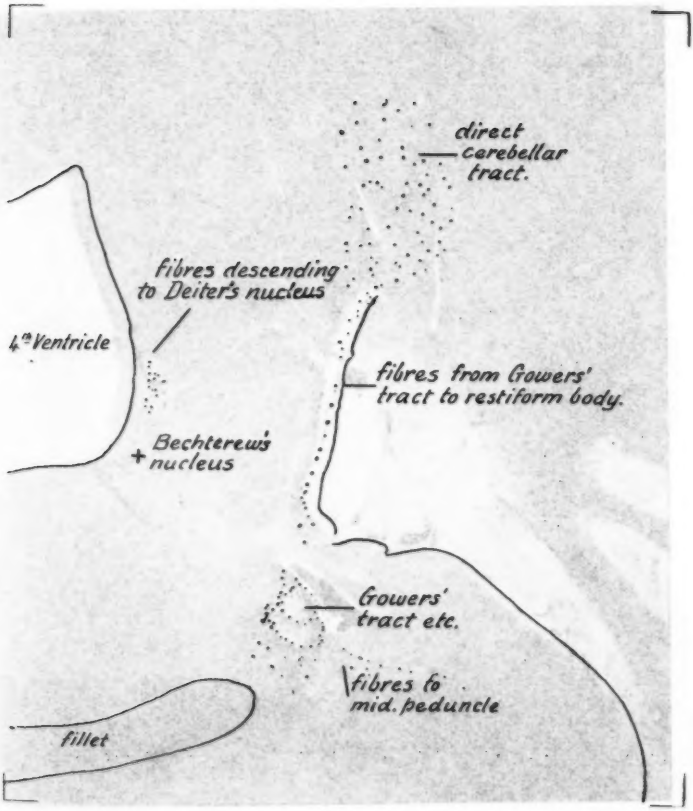

direct cerebellar tract.
fibres descending to Deiter's nucleus
4th Ventricle
fibres from Gowers' tract to restiform body.
Bechterew's + nucleus
Gowers' tract etc.
fibres to mid. peduncle
fillet

PLATE IX.

FIG. 1.

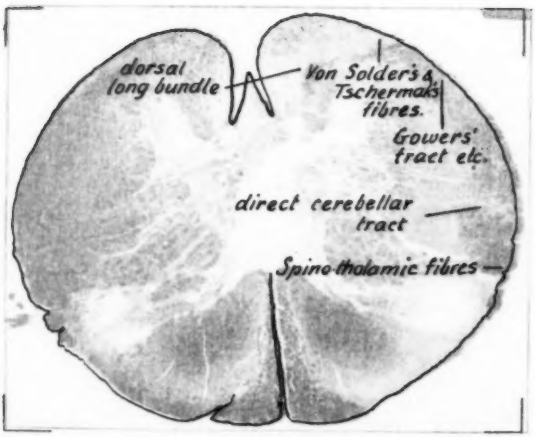


FIG. 2.

lat. int. angle of
4th Ventricle
nucleus
+ cereb. ocust.
fibres
descending
to Deiters n.
fibres entering
+ Bechterew's
nucleus.

dorsal
long bundle
von Soldern &
Tschermak's
fibres.
Gowers'
tract etc.
direct cerebellar
tract
Spino-bulbaric fibres

PLATE IX.

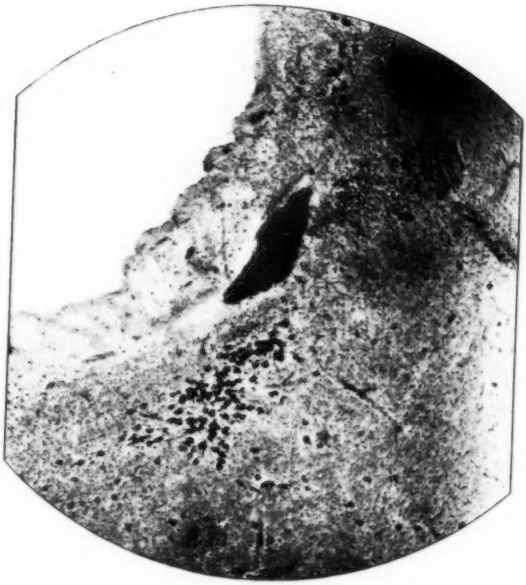


FIG. 1.

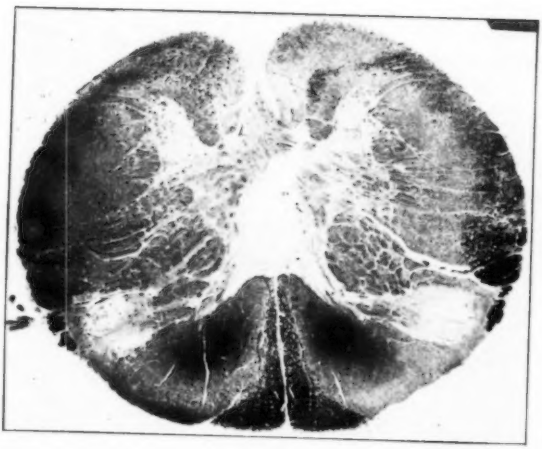


FIG. 2.

PLATE X.

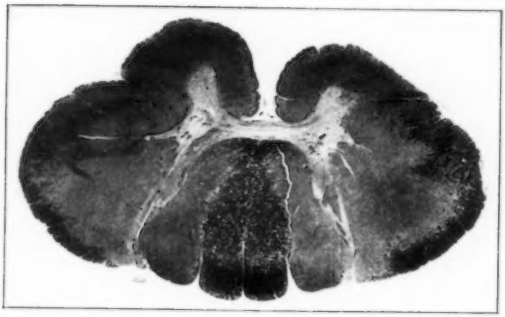


FIG. 1

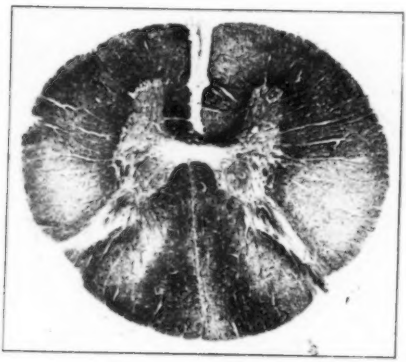


FIG. 2.

FIG. 3.

FIG. 4.

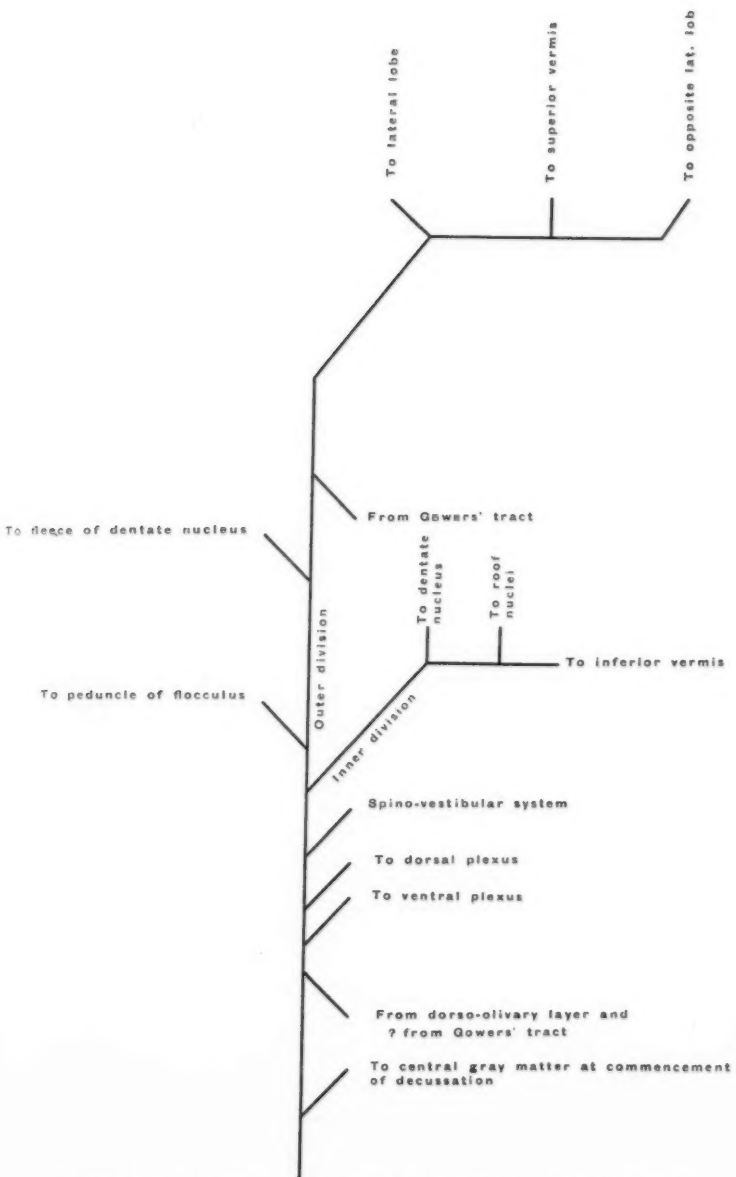


DIAGRAM OF THE LEFT DIRECT CEREBELLAR TRACT

Bale & Danielsson, Ltd., Lith.

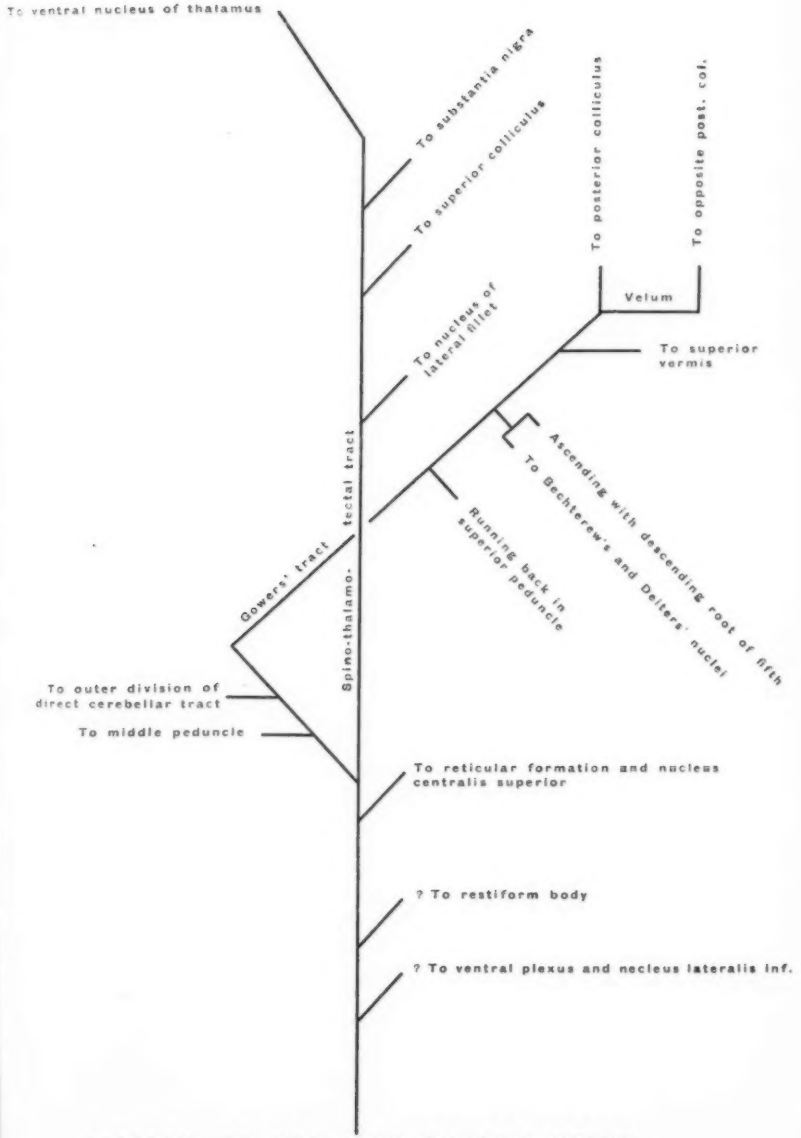
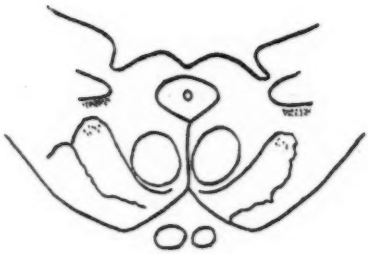
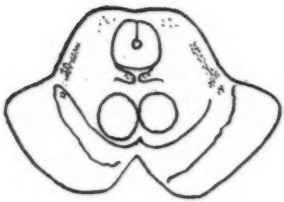

To ventral nucleus of thalamus
To substantia nigra
To superior colliculus
To posterior colliculus
To opposite post. col.
Velum
To nucleus of lateral fillet
To superior vermis
Ascending with descending root of fifth
To Bechterew's and Deiters' nuclei
Running back in superior peduncle
Gowers' tract
Spino-thalamo-tectal tract
To outer division of direct cerebellar tract
To middle peduncle
To reticular formation and nucleus centralis superior
? To restiform body
? To ventral plexus and nucleus lateralis inf.

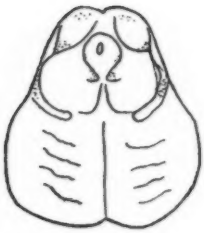
DIAGRAM OF THE LEFT GOWERS' TRACT

Bale & Danielsson, Ltd., Lith.

PLATE XI







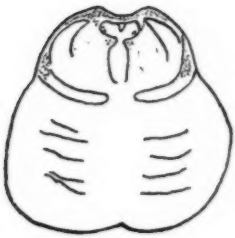


PLATE XII

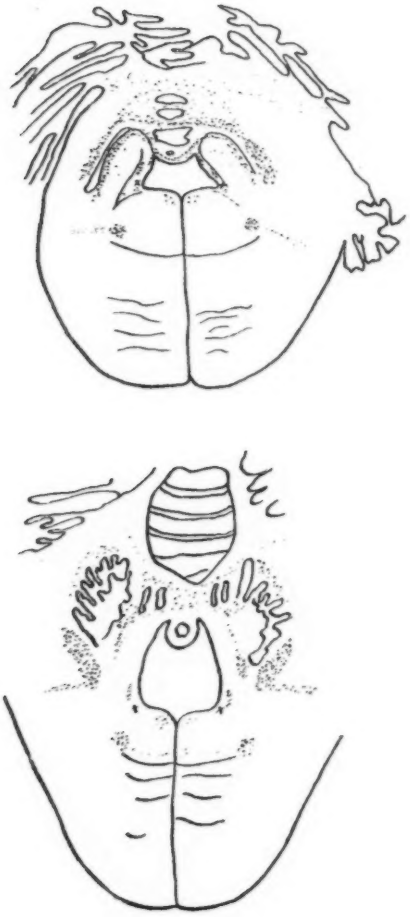


PLATE XIII

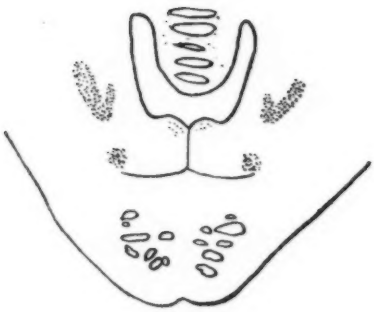


PLATE XIV

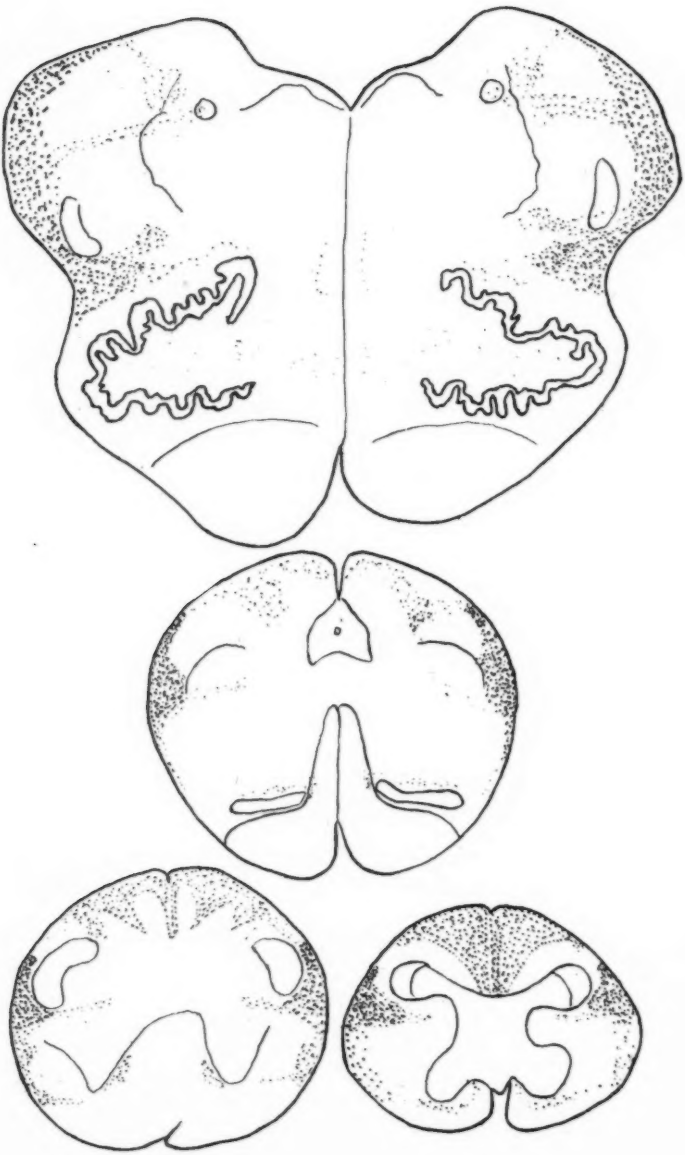


PLATE XV

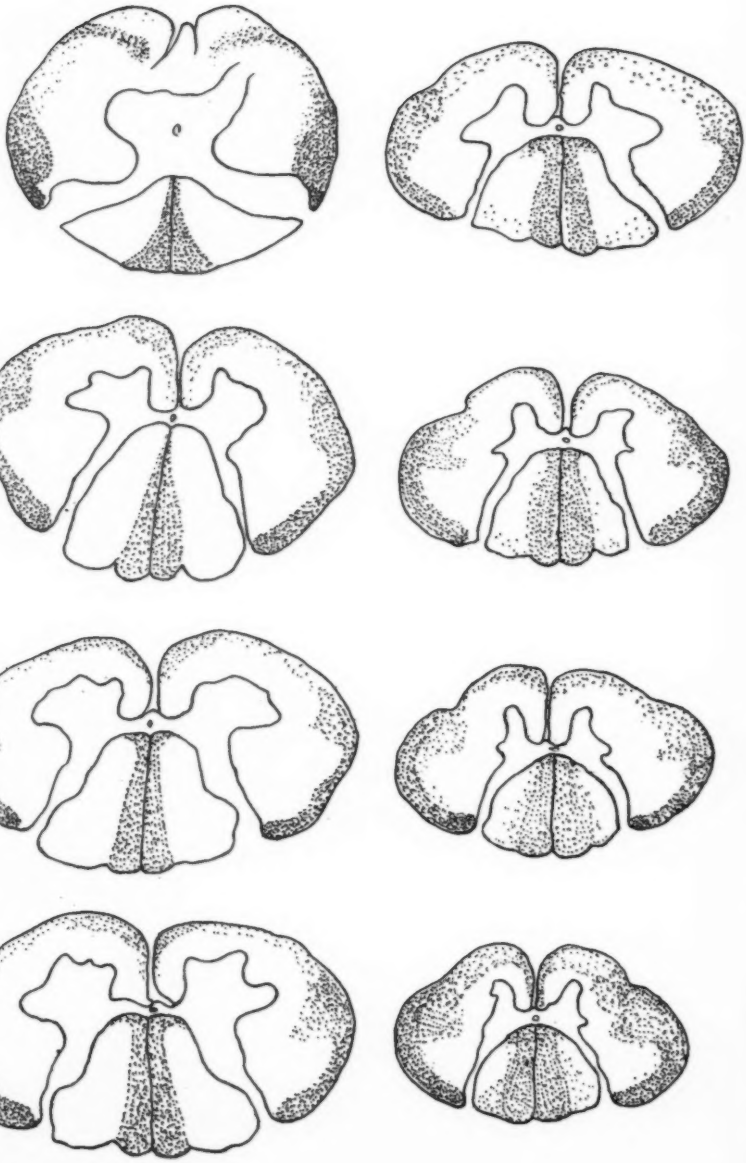
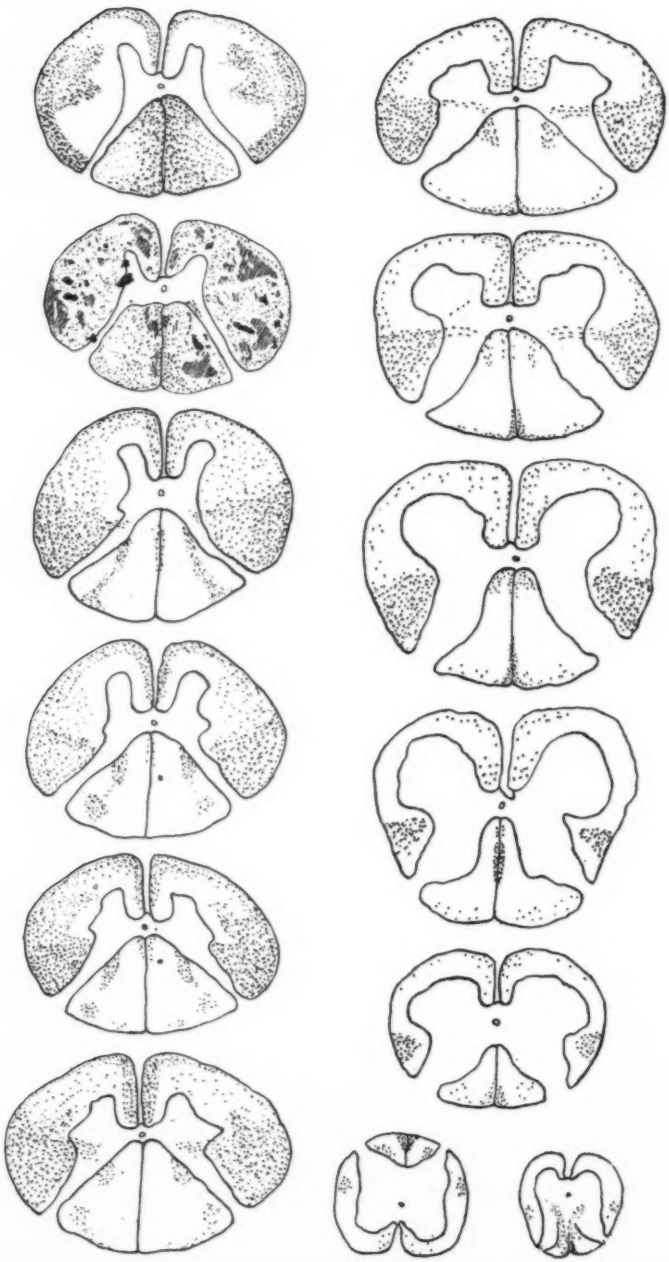


PLATE XVI



is found at successively higher levels above a transverse lesion, yet we cannot confirm the very common statement that the bulk of the degeneration in Gowers' tract cannot be traced above the cervical region, and we suggest that the earlier disappearance of the degenerated myelin from the more distal parts of degenerate neurones may account for the conflicting descriptions. We have found the position and relation of this tract in its course through the medulla and pons to be that which is usually described.

Attention may be drawn however to the very definite separation which occurs between the direct cerebellar tract, Gowers' tract, and the rest of the ventro-lateral degeneration at the level of the commencing pyramidal decussation. In the lateral funiculus of the medulla we have occasionally found the tract to occupy an unusually deep and dorsal situation lying practically between the two divisions of the antero-lateral nucleus. Fine collaterals from this tract appeared to end in these nuclei and also in connection with the ventral collateral plexus of the direct cerebellar tract.

We were unable to arrive at any definite conclusion with regard to the statement of Patrick that Gowers' tract receives an accession of fibres from the direct cerebellar tract in the lateral region of the medulla. Probably such an accession does exist, and we venture to suggest that these fibres may be those which subsequently pass from Gowers' tract at the level of the outgoing fifth nerve, to join the outer division of the direct cerebellar tract anterior to the dentato nucleus. In the lower pons we, in many cases, found the fibres of Gowers' tract arranged as a cylinder around a small mass composed of nerve-cells and non-degenerate white fibres, but no collaterals entered this mass.

From the mesial aspect of the tract a considerable number of collaterals were given off to the reticular formation, some of them passing inwards to reach the nucleus centralis medius.

No collaterals could be traced to the superior olive, nor directly from this region to Deiters' and Bechterew's nucleus.

At the level of the fifth nerve and just before the separation of the spino-tectal and spino-thalamic systems from

their association with Gowers' tract, we constantly found a strong bundle of fibres given off from the outer side of the tract, which was directed outwards, downwards, and somewhat backwards in the anterior portion of the pons, whence they run to the lateral lobe of the cerebellum *viâ* the middle peduncle.

Just before the lateral surface of the brachium conjunctivum becomes free, a strong bundle of rather coarse fibres left Gowers' tract to pass outwards and to join the outer division of the direct cerebellar tract just in front of the dentate nucleus. This bundle corresponds with that described by Auerbach, but we were unable to follow it as he described, on a path mesial to the dentate nucleus and ending in the latter body. It seemed definitely to join the outer division of the direct cerebellar tract.

Just posterior to the posterior colliculus, and as it is lying immediately ventro-mesial to the mesencephalic trigeminal root, Gowers' tract separated from the spino-tectal and spino-thalamic systems and passing obliquely upwards and backwards traversed the gray layer covering the superior peduncle and entered the velum, many of the fibres crossing to the opposite side, most of the fibres apparently ending in the superior vermis.

From the velum fibres passed conspicuously to the posterior colliculus of both sides by way of the frænulum. A certain number of fibres also turned back from the velum at the level of the decussation of the fourth nerve to run backwards in the superior peduncle.

We have now to draw attention to our observations upon an important question which has been raised by Horsley and Thiele, in respect to certain fibres descending from the velum anterius to the region of Deiters' nucleus and lying in the gray matter mesial to the brachium conjunctivum.

Those authors found these fibres degenerate in a transverse lesion of the spinal cord. They conclude that these fibres are derived from the opposite Gowers' tract, and that they are identical with the system of fibres so conspicuous in Weigert sections of the fœtal brain stem, which descend from the region of the root nuclei to Deiters' nucleus and which

has been described by Stilling, Bruce, Edinger, Kölliker, and others, and which are described as having their nuclei of origin in the nuclei tecti.

Further, Horsley has in the cat traced degenerate fibres from Gowers' tract across the root of the fourth ventricle into the gray matter lying between the brachium conjunctivum and the lateral wall of the ventricle.

In all our cases we have found degenerate fibres from Gowers' tract to Deiters' nucleus, which we describe as follows:—

In sections immediately posterior to the posterior colliculus, where the dorsal edge of the brachium conjunctivum is sinking, and where the upper part of the nucleus cerebello-acusticus is in close proximity with the dorsal fibres of the brachium, numerous degenerate fibres of Gowers' tract, certainly of the same side, and possibly also from the opposite side, were seen to turn sharply backwards and ventralwards and pass between the mesial aspect of the brachium and the nucleus cerebello-acusticus, here mingling with the fibres of the descending root of the trigeminus. In all posterior sections these fibres were conspicuous, lying first dorso-lateral to the nucleus cerebello-acusticus, then dorso-lateral to Bechterew's nucleus. These fibres could be seen constantly entering and breaking up in these nuclei, gradually diminishing in numbers, being lost below the upper part of Deiters' nucleus.

All along the lateral edge of the velum degenerate fibres could be traced from Gowers' tract into the gray matter between the brachium and the wall of the ventricle, but we were unable to trace such fibres for any distance.

It is of some importance to note that as the fibres from Gowers' tract to Deiters' nucleus turn down and back among the fibres of the descending root of the fifth, a few ascending fibres following this root were traceable in rapidly diminishing numbers as far as the superior colliculus, where they appeared to terminate in the nucleus lateralis superior.

While our conclusions confirm those of Horsley and Thiele as regards fibres derived from Gowers' tract passing to Bechterew's and Deiters' nuclei, we think that these

fibres are entirely distinct from that system described as passing from the nuclei tecti to Deiters' nuclei for the following reasons. We have examined a case of abscess limited to the root nuclei and have traced the degeneration in the descending systems of fibres passing to Deiters' nucleus (BRAIN, 1901). The plane in which these fibres run is almost a vertical plane from the root nuclei, whereas the plane of the fibres from Gowers' tract is very oblique. Again, we would point out that the point of separation of the latter fibres from Gowers' tract is comparatively distant from the root nuclei.

Lastly, the relation of the degenerate fibres to Deiters' nucleus was quite different in the case of the lesion of the root nuclei that we have mentioned.

The Spino-tectal and Spino-thalamic Tracts.

When the separation of Gowers' tract occurs these systems lie as a compact bundle of fibres ventro-mesial to the descending root of the fifth nerve. As the lateral fillet rises to cover in the brachium conjunctivum laterally, the fibres lie in that structure. Immediately behind the nucleus of the lateral fillet fine fibres arise in considerable numbers from the mesial aspect of the tracts under consideration, which pass on to the mesial side of the nucleus of the lateral fillet and end in that nucleus.

The tract now divides into a ventral and a dorsal portion; the ventral portion (spino-thalamic fibres) remains in the region of the fillet close to the lateral sulcus, while the dorsal portion (spino-tectal fibres) rises towards the oval nucleus and runs in the cup of white substance in which that egg-shaped nucleus rests. Some of these fibres end in the oval nucleus.

From the posterior colliculus the spino-tectal tract extends forwards into the superior colliculus, occupying a roughly oval area closely ventro-mesial to the commencement of the brachium posterior, and between the latter structure and the deep white fibres.

Many fibres end here in the nucleus of the superior colliculus and in the deep gray matter, and some cross in

the tectal commissure to end in a similar position upon the opposite side. The fibres occupying the fusiform area above described do not all end in the colliculus, but after all the fibres to the colliculus had been given off they appeared to shift down and out to a position roughly one-third nearer the lateral nucleus than the dorsal surface of the tectum and close to the lateral surface, where they were joined by the chief bundle of spino-thalamic fibres, which we spoke of as retaining their position in the region of the lateral fillet when the spino-tectal bundle was given off.

As a matter of fact the tendency of the fibres to sink ventralwards, as seen in transverse sections, was apparent only, and was due to the diminution of the height of the tectum. Towards the anterior parts of the superior colliculus the spino-thalamic bundle sunk rapidly to touch the substantia nigra, to which many collaterals were given off. Coming here in contact with the mesial surface of the internal geniculate body it swung abruptly outwards in close contact with the under surface of that structure into the ventral nucleus of the thalamus, beyond which we have failed to trace it. No collaterals were given off to either geniculate body. Referring to the publications of Rossolimo and of Troschin upon this subject, we would draw attention to the fact that both these authors described fibres from this tract entering the substantia nigra at the lateral angle, which could be traced across the breadth of this structure as far as the middle line. We have been able to trace the fibres only into the outer fourth of the substantia nigra.

Further, Rossolimo is quite certain, both in his statements and figures, that in his cases the fibres cross the ventral nucleus of the thalamus and end in the globus pallidus of the lentiform nucleus.

We have failed, however, to follow them farther than the ventral nucleus.

The Ventral Restiformal Spino-cerebellar System of Tschermak.

We have found a variable number of fibres passing from the ventro-lateral region of the first cervical segment to the

medulla which seem to correspond to the fibres described by Tschermak.

In the first cervical segment these fibres were situated in front of Gowers' tract and passed inwards, following the course of the ascending fibres of the dorsal longitudinal bundle to lie deeply immediately external to the pyramid; the fibres were here mixed with von Solder's fibres and the fibres of the dorsal longitudinal bundle. At the commencement of the pole of the olive some of these fibres passed directly into the dorso-olivary layer, while others passed first mesial to the olive and then turned into the dorso-olivary layer. These fibres passed outwards and appeared to join the direct cerebellar tract by looping under the spinal trigeminal root. We found the largest number of these fibres in two cases of subacute combined degeneration in which the lesion of the ventro-lateral region extended as high as the third cervical segment.

von Solder's fibres were present in variable numbers in some cases, but in several cases of quite recent degeneration we failed to demonstrate them.

They pass from the ventro-lateral region of the first cervical segment with the fibres of the dorsal longitudinal bundle and Tschermak's fibres to the mesial side of the inferior olive, from which region a few fibres entered the olive; they were then continued upwards in the outer part of the middle fillet, and appeared to join the spino-thalamic degeneration at the knee of the lateral fillet.

Their inconstancy suggests they may be fibres of the spino-thalamic system taking an unusual course.

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Reviews.

Die Geschwülste des Gehirns, von Professor Dr. OPPENHEIM in Berlin. 2te, erweiterte Auflage. (Alfred Hölder, Vienna, 1902.)

INCREASING knowledge of the functions of the various parts of the brain has led to a correspondingly greater exactitude in the diagnosis of intracranial lesions, and the prognosis has thereby in many cases become materially improved. The value of a text-book such as that of Professor Oppenheim, dealing with brain tumours, is therefore a high one, and in this new edition we find a succinct account of our present-day knowledge on the subject.

Commencing with a description of the pathological anatomy and histology of intracranial growths, Oppenheim discards as unpractical any rigid classification, whether based on grounds of histology, embryology, starting-point, or direction of growth, and prefers to consider each variety of tumour separately.

The various forms of new growth in the brain, meninges and cranial bones are fully described, and an interesting section follows in which is discussed the influence of intracranial growths upon the brain, the cerebral ventricles and the cranial nerves. Local softening, which not infrequently occurs in the vicinity of a tumour, is sometimes inflammatory in origin, due to an encephalitis; in other instances it is the result of compression and occlusion of neighbouring vessels. Internal hydrocephalus may be produced either by venous stasis, the arteries remaining pervious, or by direct pressure on the channels of communication between the cerebral ventricles.

The general etiology of intracranial new growths (apart from developmental, infective and parasitic forms), is briefly discussed. On the question of a possible association between local trauma and a subsequently forming neoplasm, he points out that the antecedent head injuries described in certain cases are sometimes the result of vertigo or other sudden cerebral symptom, which latter may in its turn be due to a pre-existing cerebral tumour. In other cases, a trauma serves merely to excite more

rapid growth in a latent tumour. But, in spite of such cases, he admits that all post-traumatic cases cannot be so accounted for, notably instances of tumours forming in the site of old scars in the bones or meninges.

The great mass of the book is devoted to the description of the symptomatology of intracranial growths and their focal diagnosis. The care with which this is done cannot be too highly commended. There is also a short discussion as to the pathological diagnosis of the nature of a tumour, apart from its anatomical position. Special attention is drawn to the multiform clinical appearances which may be met with in cases of hydatid cysts.

As to treatment, he recalls Wernicke's dictum as to the value of massive doses of potassium iodide, even in non-syphilitic growths. Thus sarcomata, gliomata and tuberculous tumours are sometimes favourably influenced by this drug. Oppenheim himself inclines to think that the cystic tumours, cystic gliomata and sarcomata, are those which best respond to potassium iodide, but tend to be aggravated by mercury. The iodide should be commenced in moderate doses and pushed boldly to large quantities.

The surgical treatment of brain tumours is carefully considered, and it is gratifying to the British reader to find the generous manner, all too rare in German text-books, in which the work of English and American observers is acknowledged.

von Bergmann's statistics of 285 cases of cerebral tumour treated by operation are of great interest, and their main points may here be recapitulated. In 116 cases the tumour was correctly localised and reached, whilst in 157 operation was undertaken for exploratory or for palliative purposes. Of the 116 cases of radical operation, 29 (or 25 per cent.) died immediately after operation, whilst 87 (75 per cent.) survived. In 60 (51·59 per cent.) improvement or cure resulted. Of the 157 cases in which the cranium was opened without extirpating the tumour, in 13 the operation could not be completed owing to hæmorrhage, shock, &c. In 16 no tumour was present. In 89 (56·65 per cent.) it was present in a different situation from where it had been expected, and in 43 it was either too large or too diffuse for removal. Only in 14 cases of the 157 was the operation deliberately undertaken solely for palliative purposes. The mortality of the 157 unlocalised cases was 75 or 47·77 per cent. Thus the combined mortality of the whole 278 cases was 104, *i.e.*, 36·4 per cent. Allowing for other unsuccessful, and therefore pro-

bably unpublished, cases, Oppenheim considers that this figure rather underestimates the actual mortality. He has since added to Bergmann's statistics those of cases collected by himself from the literature, and the following are the results so obtained:—Out of 371 cases in which the cranium was opened with the diagnosis of intracranial tumour, 140 or 37·7 per cent. died within a day or two of the operation. Eighty-eight cases, 23 to 24 per cent., were cured, or appreciably improved. In 111, or 29 to 30 per cent., only transient improvement resulted. This includes some of the palliative operations, and also those in which the tumour was either not found or only partially extirpated, or in which transient improvement followed the draining of a cyst, while the actual tumour was not removed. In 166 radical operations for correctly localised tumour 82, or nearly 50 per cent., were cured or distinctly improved.

It must always be borne in mind that even successful removal of a tumour can only relieve the general intracranial symptoms, such as headache, vomiting, vertigo and optic neuritis, but cannot restore local paralytic phenomena due to destruction of brain tissue, though a certain slight amount of compensation by surviving areas may take place.

Opening of the cranium and dura mater, whether the actual tumour be accessible or not, generally relieves optic neuritis. The improvement sometimes begins within twenty-four to forty-eight hours after operation. The other general symptoms, headache, vomiting, giddiness, mental dulness, &c., also improve within the first few days in favourable cases, whilst focal symptoms take much longer to become compensated.

Next to fibromata, lipomata, or other simple tumours, those which offer the best prospect of radical cure are the sarcomata, though favourable results are sometimes attained with gliomata, solitary tuberculous masses, patches of tuberculous meningo-encephalitis, angiomata, gummata and parasitic cysts.

He quotes *in extenso* Sir Victor Horsley's opinion as to the advisability of operation at the earliest possible date after a tumour has been diagnosed, and as to the serious responsibility of waiting till the patient is half blind from optic neuritis or atrophy, or until the tumour has increased to such an extent as to greatly increase the risks of its removal.

In cases of inoperable tumour, Oppenheim refers to the plan of repeated withdrawal of cerebrospinal fluid by lumbar puncture for the purpose of relieving intracranial pressure, but only approves of this in cases where the symptoms point to interna

hydrocephalus, such, for example, as tumours in the posterior fossa of the brain.

It is difficult, within the limits of a short review, to give an adequate account of the wealth of material in this book, or of the exactitude with which it has been compiled.

PURVES STEWART.

Handbuch der pathologischen Anatomie des Nervensystems. Edited by Dr. E. FLATAU, Dr. L. JACOBSON, and Dr. L. MINOR. (Published by Williams and Norgate, 14, Henrietta Street, Covent Garden, London.)

THE first volume of this work has already been reviewed in the summer number of this journal, and the work is now complete in four additional numbers. The first chapter of the second volume is written by Dr. H. Stroebe, and deals with the changes found in the skull and membranes of the brain. The pathology of chondrodystrophia fœtalis, rickets, osteomalacia, osteitis deformans, leontiasis ossea and acromegaly are all dealt with. The various views held with regard to the formation of hæmorrhagic pachymeningitis interna are discussed at some length.

Tuberculosis of the skull and membranes is described, and it is stated that a tuberculous osteitis frequently occurs in children in the supraorbital region, and leads to perforation of the bone and to a tuberculous pachymeningitis externa, these perforations being not uncommonly multiple.

The syphilitic affections of the skull and membranes are next described. Actinomycosis of the base of the skull and the membranes arising from carious teeth or other foci in the mouth or jaw is a rare affection, but cases have been described in which the dura mater has been perforated, and the pia mater and the brain substance invaded.

In discussing the pathology of epidemic cerebrospinal meningitis the view is taken that the disease is not necessarily due to one specific organism, such as that described by Weichselbaum, but may be due to a variety of organisms. In acute leptomeningitis there is almost always an inflammatory œdema of the brain, with thrombosis of the smaller vessels. Chronic leptomeningitis most frequently arises in connection with some disease of the neighbouring structures, but may be found in chronic lead

and alcohol poisoning and in chronic kidney disease. Tuberculosis of the pia mater is, again, almost always a tuberculous meningo-encephalitis with œdema of the brain substance.

The developmental defects of the brain are described by Professor G. Anton, who also writes the chapter on œdema of the brain and compression.

After describing cases of anencephaly, the non-development of the basal ganglia, the so-called cyclops brain, and other forms of defective development, he deals with the condition known as porencephaly, and gives tables showing that the motor region of the brain is that which is most commonly affected.

The chapter on microcephaly contains diagrams and tables of comparative weight of the brains in such cases, and according to the weights they are divided into three classes. The causes of microcephaly are discussed, and the view is taken that the smallness of the vessels and the premature synostosis of the skull are probably secondary to the non-development of the brain and not the cause. The undue contraction of the uterus, injury during birth, and maternal influences, are mentioned as possible causes. The condition of the amnion is also an important factor as well as alcohol and syphilis in the parents.

The next chapter, written by Dr. M. Friedmann, deals with hyperæmia, anæmia, hæmorrhage, embolism, thrombosis, encephalitis and abscess of the brain, and a very good set of illustrations is given showing the appearances met with under these conditions.

The next chapter, by Dr. L. Bruns, on cerebral tumour and parasites in the brain, is possibly one of the most important. He points out that nearly 64 per cent. of all cases are of the nature of glioma or sarcoma. When, however, cases of cerebral tumour occurring in children are included in the figures the percentage is less, owing to the greater number of tuberculous cases. The distinction between gliomatous and sarcomatous tumours is based partly on macroscopical, partly on microscopical characters. The sarcomatous tumours are sharply defined from the brain substance, whereas the gliomatous invade the brain substance, and no sharp line of separation can be seen between healthy brain tissue and tumour substance. Stroebe states that by the character of the cells on microscopical examination it is possible to distinguish between the two conditions. The word "glio-sarcoma" should only be used for cases in which there is a true combination of the two conditions. The conclusions arrived at after a careful consideration of the subject are :—

(1) That a glioma is histologically and histogenetically distinct from a sarcoma.

(2) That a glioma is an infiltrating tumour, and it is impossible to distinguish between the connective tissue which forms the glioma and that which forms the brain substance.

(3) A glioma may contain numerous nerve-fibres and nerve-cells which retain their function, and hence the disturbance in function is often less than might be expected from the situation and extent of the lesion.

(4) Large hæmorrhages in glioma are frequent, and may give rise to severe apoplectiform symptoms in apparently healthy individuals, owing to the fact mentioned above, viz., that cerebral functions are not much disturbed by the growth.

The rarer forms of tumour, the cholesteatoma and dermoid, are described. The rarity of primary carcinoma of the brain is pointed out, and it is stated that the majority of cases of true carcinoma of the brain are metastatic.

A very rare form of brain tumour is actinomycosis. A short chapter deals with the various parasites in the brain.

The chapter on syphilis of the nervous system is written by Professor v. Bechterew. He deals with the subject under the following headings: the gummatous growth of the central nervous system and its membranes; syphilitic pachymeningitis; syphilitic meningitis; syphilitic meningomyelitis and encephalitis; and disseminated syphilitic sclerosis.

The article on diseases of the medulla and pons is written by Dr. R Cassirer. After describing the pathology of the chronic progressive bulbar and ocular palsy, the author deals with the pathology of congenital defect of movement in the eye, the face, and tongue muscles. In these cases the affected muscle has been found to be atrophied, and on examination of the oculo-motor nucleus a very marked diminution of the number of cells has been demonstrated, and it is therefore probable that a developmental error in the nuclear region of the affected muscle is the cause of the defective movement.

The pathology of the acute and subacute affections of the medulla and pons are next dealt with, viz., polioencephalitis, superior and inferior, abscess, hæmorrhage, and softening.

The chapter on the pathology of myasthenia gravis, or "bulbär Paralyse, ohne anatomischen Befund," is written by Professor H. Oppenheim. The examination of the nervous system has revealed no abnormality. Organisms have been found in some cases, and it is known that certain organisms, such as bacillus botulinus, have the peculiarity of acting in a toxic manner on the bulbar centres. The abnormality of a

double central canal has been described, but can hardly be regarded as a factor in the disease. The observations of Weigert, who found a thymus tumour with metastatic growths in the muscle, are discussed. It is suggested that this affection is probably due to some form of intoxication.

Multiple sclerosis is shortly and concisely described by Dr. G. J. Rossolimo, while the secondary degenerations of the brain are dealt with by Professor Hocke.

The diseases of the spinal column and the membrane of the spinal cord are described by Dr. H. Stroebe. The developmental defects of the spinal cord are described by Professor Karl Petrén. Softening of the spinal cord, compressive myelitis and spinal abscess are dealt with by Professor A. Pick.

The chapter on poliomyelitis is written by Dr. A. Goldscheider and Dr. F. Brasch. In discussing the etiology of the affection mention is made of the finding of the meningococcus of Weichselbaum by Schultze and by Courmont and Bonne in the fluid obtained by lumbar puncture, and this suggests that the disease may have some etiological connection with cerebrospinal meningitis. The disease is divided into three stages: in the first the vessels are engorged, and especially the anterior central artery of the cord, and the walls and surrounding spaces are filled with numerous small, round cells. This infiltration becomes even more marked in the gray matter of the spinal cord where the vessels divide. Small and large hæmorrhages are often present. In the second stage there is degeneration and then regeneration of the affected tissue, and in the final stage all the active processes have become stationary.

Tract and system diseases of the spinal cord are described by Professor Homén (Helsingfors), and under this heading he deals with the pathology of tabes dorsalis, the cord changes which are found in dementia paralytica, the cord changes in pernicious anæmia, so-called combined degeneration of the cord, Friedreich's disease, and hereditary spastic spinal paralysis.

The secondary degenerations of the spinal cord are described by E. Flatau, and good diagrams are given of all the well-known ascending and descending tracts.

The author deals at considerable length with the question as to the origin of the descending fibres in the posterior column, in the comma tract of Schultze, in the oval area of Flechsig, and in the Gombault and Philippe bundle, and he states his opinion, based on a most careful review of numerous cases: –

(1) That the comma tract contains descending fibres derived from the posterior roots.

(2) That the dorso-median bundle in the lumbo-sacral cord has probably the same origin, viz., from fibres derived from the posterior roots.

(3) That there are in the Schultze comma tract and in the dorso-median bundle fibres which are endogenous in origin, just as there are endogenous fibres mixed up with the other long tracts of the cord.

The degeneration which takes place in the posterior roots and posterior column in cases of tumour of the brain is discussed, but the author does not express any opinion as to the origin of the degeneration.

The pathology of amyotrophic lateral sclerosis is next dealt with by Professor G. Ballet.

The traumatic diseases of the spinal cord are described by Dr. L. Minor, and a large portion of the chapter is devoted to hæmatomyelia, and he demonstrates the fact that such hæmorrhages commonly occur in the region of the posterior horns, and tend to extend in a tubular form in this region, and not to extend laterally.

Disease of the conus terminalis is dealt with by Professor Raynaud, of Paris. The subject is one which, if treated purely from a pathological point of view, would lose much of its interest, and the writer has given a considerable amount of detail, especially of the localisation of a lesion situated in the region of the cauda equina.

Of lesions in this region, 60 per cent. are due to trauma, 28 per cent. to tumours, and the remaining 12 per cent. to syphilis, meningitis, tubercle and unknown causes.

The tumours in this region consist of sarcoma, lymphangioma, neuroma, glioma and parasitic tumours. Mention is also made of the unique condition of a myolipoma of this region, described by Gowers.

Syringomyelia is described by Dr. Hermann Schlesinger, who also describes tumours of the spinal cord and their membranes.

The first part deals with *tumours of the spinal column*, of which the primary affections are almost always sarcomata, while the carcinomata are always secondary.

Innocent tumours of the spinal column are exostoses, osteomata, angioma and chondroma.

The tumours of the meninges and spinal cord are next dealt with, both malignant and innocent.

Multiple fibromata, or neurofibromata, are described, and it is stated that when these appear in the spinal canal they are

usually extensions from very disseminated tumour formation of the peripheral nervous system. These multiple neurofibroma manifest themselves in the form of smaller or greater tumours on the nerves. The cerebral and spinal nerves are likewise affected.

The cauda equina is a favourite situation for these tumours. Sometimes the tumours are present also in the spinal cord, and may in certain cases completely invade that structure.

The spinal canal may be enlarged and the intervertebral spaces widened owing to the pressure of these tumours. The multiple neurofibromata seem to have a much more unfavourable prognosis than the solitary fibroma. The average duration of the disease in a series of cases is about one and a half years.

In describing the neurofibroma of the peripheral nerves, Dr. E. Lugaro, who writes the section on the subject, says that in universal neurofibromatosis (v. Recklinghausen) or fibroma molluscum multiplex (Virchow), the fibroma are very numerous, and quite separate from one another. They are generally small, and only a few reach any noticeable size. Generally the skin nerves are affected, but the intercostal, the visceral branch of the sympathetic, the posterior roots, the anterior roots, and the spinal ganglia may be involved. Multiple fibromata in both forms, either a plexiform neuroma or a generalised fibromatosis, may be congenital or hereditary.

The question arises, what relation do the two diseases here described bear to one another? Clinically they certainly are not identical, for in the one case the disease runs its course in eighteen months, whereas in the case of multiple fibromata of the nerves the disease is of very long duration. Pathologically these two diseases may be identical—clinically they are distinct.

A very good description of the changes found in the optic nerve under various pathological conditions is given by Professor A. Elschnig, of Vienna; but little is said with regard to the direct effect of vascular disease on the optic nerve.

The chapter on the pathological condition of muscle is written by Professor L. Darkschewitsch, and much valuable material has been collected, but a considerable portion of the chapter is devoted to clinical observations which, although interesting in themselves, might well have been omitted from a work devoted to pathology. It falls to the lot of no one man to be able to investigate the pathology of the numerous morbid conditions of muscles which exist, firstly on account of the chronicity of the affection, and secondly on account of their rarity in omparison with other diseases, and therefore a work on athological

muscular condition must to a large extent be a compilation of the work of others. In the present article, however, the observations are to a considerable extent the author's own, and especially in regard to muscular atrophy in association with joint disease, subacute poliomyelitis and the especially interesting form of muscular atrophy which occurs in cerebral disease.

The last volume deals with the pathology of epilepsy and eclampsia, tetanus, tetany, chorea, paralysis agitans, and exophthalmic goitre.

Two interesting chapters follow on the disease of the skin and the closely-related changes in the bones and joints in nervous affections.

The pathology of the pituitary body is written by Professor Benda, of Berlin, and that on deformity of the skull by Dr. Weber.

The final chapter on the pathology of the psychoses is written by Dr. A. Cramer, of Göttingen.

In the introduction published with this last volume the collaborators point out the extreme importance of a knowledge of the pathological anatomy in understanding the diseases of the nervous system.

Much in nervous pathology is even yet not clear and hard to explain. The comparative recent development of knowledge of nervous pathology accounts for the absence of any complete book on the subject, while there are numerous excellent works on the normal anatomy of the nervous system.

The need for such a work as the present is undoubted, for it may be stated that there is no work which covers the ground taken up by this book.

Mistakes may be present, but since each chapter is written by an author who has devoted special attention to the subject on which he writes, the chance of error is rendered very small.

The Editors issue this work to the medical world in the hope that it may form a basis for the further development of pathological anatomy.

It is impossible in a review of a work of this nature to do justice to the many excellent articles which it contains, but those who are investigating the pathology of the nervous system will find it an invaluable book of reference.

FREDERICK E. BATTEN.

Hypnotism, its history, practice and theory. By J. MILNE BRAMWELL, M.B., C.M. (London: Grant Richards, 1903, pp. 478.)

THE pages of BRAIN have been indebted to Dr. Bramwell from time to time for articles, reviews and critical digests bearing on hypnotism, and his systematic treatise is sure to find a large circle of readers. The subject is mysterious, bordering on the occult, and many have been deterred from giving it due consideration because, in the past, many of its exponents have made extravagant claims as to its therapeutical value, and because, in some cases, deception has played an important part in its supposed manifestations. It is, therefore, a great advantage to have a treatise like the one under consideration, which is written by one who has had long personal experience and in which the different sides of the question are treated with fulness and in a fair and scientific spirit.

The historical section is written in an interesting manner although its purview is rather partial. Consideration of Mesmer and his followers is purposely omitted, the reader being referred to the treatise of Moll. There is a good biographical notice of Elliotson and of his somewhat stormy career. It is rather surprising to find the late Herbert Spencer among the contributors to Elliotson's heretical journal, *The Zoist*. He contributed articles in the early fifties on "A New View of the Functions of Imitation and Benevolence"; "On the Situation of the Organ of Amativeness"; "A Theory Concerning the Organ of Wonder." Dr. Bramwell also gives an excellent account of the work of Esdaile in India. Esdaile used hypnotism largely as an anæsthetic in surgical practice, and it was some experiments of Esdaile which the author witnessed when a boy that aroused his own interest in the subject. There is also a sketch of the work of James Braid, the Manchester surgeon, to whom we owe the use of the word hypnotism. Each of the three was an enthusiast fully convinced of the paramount importance of his pet subject. They had to struggle against the scepticism and conservatism of their colleagues, and submit to accusations of credulity or worse, and in return they carried the war into the enemy's country with an energy, and often a lack of tact, which greatly impeded them in their attempt to induce the profession to take their views into serious consideration. The allusions to Liébault and other subsequent investigators seem rather meagre compared with the space given to the three British pioneers.

Dr. Bramwell discusses the various methods which have been employed to induce and to arrest hypnosis. He himself relies very little on mechanical aids such as passes or revolving mirrors, but endeavours to get the patient into an expectant state of mind, and to induce hypnosis mainly by suggestion. In many cases he employs simultaneously suggestions intended to induce hypnosis with others intended to cure or relieve disease, and states that "frequently the latter is successful before the patient can be described as genuinely hypnotised." To dehypnotise the patient, Dr. Bramwell prefers "to *suggest* during hypnosis that the patient shall awake at a given signal, as, for example, when the operator utters the word 'awake' or counts 'one two three.'"

The chapter on the experimental phenomena of hypnotism, occupying some eighty pages, is of great scientific interest. The phenomena of hypnotism, as Dr. Bramwell has observed them, are very well and clearly described. He throws doubt on some of the more marvellous, such as the elevation of blisters by suggestion when pieces of plain paper are applied, as fraud does not seem to have been satisfactorily excluded. Post-hypnotic phenomena are dealt with very fully. There is in particular an extremely interesting account of observations on a subject in whom post-hypnotic appreciation of time was developed to a remarkable degree. She was told, when hypnotised, that at some given number of minutes afterwards she was to get a piece of paper and mark a cross on it. The only direction given was the number of minutes, no calculation being made at the time as to the hour when that would fall due. In nearly every case the suggestion was acted on with absolute accuracy, and in others where the number was very large, *e.g.*, 20,180, she was only one or two minutes out. Cases are also detailed showing a continuity of memory from one hypnotic period to the next, although during the waking state the subject had no recollection of anything which had taken place during the hypnotic stage. The experiments mentioned in this chapter are described in full detail, and every care seems to have been taken to exclude error or deception.

A hundred and twenty pages are devoted to an exposition of Dr. Bramwell's views on the value of hypnotism in medicine and surgery. He quotes many cases from his own experience in which painful short operations, such as extraction of teeth or avulsion of toe-nails, were performed without suffering, and quotes from other authors instances of painless parturition under hypnotism. But he finds that sufficiently deep anæsthesia can only be induced in a small minority even of those who are

susceptible to hypnotism. In the treatment of medical affections most benefit is claimed in various forms of hysteria, in the removal of obsessions and imperative ideas, and in various neuralgic affections, such as dysmenorrhœa. In insanity only three out of eight cases proved susceptible, and in them the effect of treatment was not very evident. Dr. Bramwell gives an analysis of seventy-six cases of chronic alcoholism which he has treated by hypnotic suggestion. Of these twenty-eight recovered, and thirty-six "improved." Of the twenty-eight who recovered, seventeen were males, and eleven females; the average age was 40; the average number of times of treatment was thirty, and the average length of time which has elapsed since recovery three years. In many of the medical cases described in this chapter it is difficult, one must admit, to feel certain that hypnotism had any more specific effect on the improvement than any other new and impressive mode of treatment might have had. The personal influence of the operator, apart from hypnotism, is clearly a large factor. This is confirmed by Dr. Bramwell's statement on p. 422, that "out of fifty-nine illustrative *medical* cases, twenty-eight were hypnotised, and thirty-one presented no symptoms of hypnosis, *i.e.*, despite the fact that these patients all underwent hypnotic treatment, none of them presented *unmistakable* symptoms of hypnosis. Further, though many of the latter were cases of grave and longstanding illness, yet in some instances the recovery was extremely rapid."

With reference to the supposed dangers of hypnotism Dr. Bramwell is very optimistic and emphatic. Objection has been taken to its employment on the ground that it might do mental or physical harm to the patient, or that he might be liable to danger from being unable to resist improper suggestions made by the operator. Dr. Bramwell considers that these fears are void of foundation, and declares (p. 425): "Personally I have never seen a single hypnotic somnambule who did not both possess and exercise the power of resisting suggestions contrary to his moral sense." It is, however, difficult to reconcile this with Dr. Bramwell's account on p. 317 of an experiment which he performed before the York Medical Society: "I was requested to give an example of changed personality by making this subject (a shopkeeper, aged 25) believe that he was a dissenting minister preaching a sermon. He refused to do this, and I was then asked to make him believe he was a hawker selling fish. This was also rejected, but he accepted the suggestion that he was Barnum, and that the medical men were wild beasts, and pro-

ceeded to describe them in a highly amusing manner. I afterwards tried to make him accept the first two suggestions, but invariably failed. On one occasion, however, he accepted the suggestion that he should poison a personal friend. The subject at that time was in the 'alert' stage of hypnosis, with his eyes open. I took a lump of sugar from the basin and assured him him that it was a piece of arsenic sufficient to kill a dozen persons. I then put it in a cup of tea and told him to give it to his friend to drink. He did so at once. I asked him why he had poisoned his friend, and he replied laughingly, but in an unnaturally gruff voice, 'Oh, he has lived long enough . . . . . .' He affords the only instance in which an imaginary crime has been carried out by one of my own subjects."

One-third of the book is taken up by a critical account of the various theories which have been advanced to account for the phenomena of hypnotism. They are given very fairly and fully, but the discussion on them is often discursive, and would bear much excision and condensation.

The value of the work is increased by an exhaustive bibliography and copious indices, and the printing is excellent.

The book is thoroughly worthy of study as a fair and candid presentation of the views of the author on a subject about which he is entitled to speak with authority both as an investigator and a practitioner. The value of future editions would be increased if some redundant passages and repetitions were omitted, and the subject matter in some places rearranged, *e.g.*, the theoretical parts, which are at the beginning and end of the work, might very well be incorporated with one another.

W. S. COLMAN.

Pathologie mentale des Rois de France, Louis XI. et ses ascendants, une vie humaine étudiée à travers six siècles d'hérédité, 852-1483. Par AUGUSTE BRACHET. (Paris, Hachette, 1903, royal octavo, pp. 913. Prix 15 fr. net.)

THIS learned work was begun in 1880 on the advice of M. Littré, to whom it is dedicated. It was printed in 1896 for private circulation in four volumes, to which the Academy of Medicine accorded an honourable mention. Death overtook the author before he had quite finished the work for the present

edition. The volume is edited by his widow, Madame Anna Brachet.

The author commences by stating that the clinical history of humanity can only be written from the records of royal dynasties, or of families so illustrious that their ancestors and collateral relations are known. He sets little value upon studies of "sporadic" distinguished men, because we know little or nothing about their ancestry.

The period which M. Brachet covers by his study comprises twenty generations. With a learning and diligence beyond praise he has gathered together all the records that time has left us about the ailments of these kings and princes and their wives, and the ancestors of their wives. This task, sufficiently laborious, would have been too great, even for M. Brachet, were it not for the frequent consanguineous marriages, which considerably reduce the number of ascendants.

The introduction of 219 pages which should contain the gist of M. Brachet's researches had to be completed from notes left by him. The remaining 694 pages contain a series of extracts, chronologically arranged, about the diseases of the French kings from St. Louis to Charles VI., who was insane. Here there is an unfortunate break, for it is very doubtful whether Charles VII. was the son of the mad king. M. Brachet leaps over this barrier by showing that Louis XI. came from the house of Anjou by his mother. M. Brachet would make out that king to have been insane before he died on grounds that do not seem quite satisfactory. We are not sure whether there was any decided neurosis in the royal dynasty of France, for insanity may occur without being inherited, and families in which it is inherited sometimes wear out the ancestral taint. One thing greatly needed in researches of this kind is a scale to let us know what is the proportion of nervous or other hereditary diseases in ordinary families. We should then be more able to inquire whether such diseases are more frequent in royal families. We think that insanity has appeared in all European royal families save the Stuarts.

The learned author devoted five pages to show the uselessness of the work of the alienist, Ireland, who has bestowed only fifteen pages on the family of the Emperor Charles V. without ever consulting the sources of information, whereas the only scientific way of getting physiological conclusions is the detailed study of the texts. Upon this footing M. Brachet assures us the dynasty of Charles V. would demand, with the preparatory pathological

biography of each personage based upon the originals, three volumes and ten years of work. "Twenty years are needed for what I attempted to do for the French dynasties." Clearly Ireland's duty was to have passed by the books of Merimée, Mignet, Bergenroth, Stirling, Gachard, and others, for the documents upon which these painstaking monographs were written; he should have passed months in Simancas and in the Musée des Chartes, and in default of sundry continental libraries spent hundreds of pounds upon rare books and pamphlets. Then, instead of half a dozen sketches of fifteen pages each, he could, at the end of a long life, have bequeathed to the world a score of volumes weighing about five stones upon the subjects which he has outlined. When this alienist's end draws nigh how will he deplore such neglect, like the scholar who regretted that he had not devoted his life to the dative case.

It may be further observed that though M. Brachet pronounces Ireland's historical studies on insanity to be baseless he nowhere tries to show their incorrectness, except when he comments upon his observation that after much inquiry he could find no support for the assertion that insanity was rife in the house of Burgundy, save that the mind of Charles the Bold seems to have become unhinged by the defeats which he suffered from the Swiss. Against this Brachet brings the Milanese ambassador, who wrote that the Charles changed counsel every day and was mad (matto), though he admits that Philippe de Commines merely said that he had lost much sense (beaucoup diminué de sens).

The grandfather of Charles the Bold had gout, and his father had apoplexy. Here we have to settle with Dr. Haig whether gout be a neurosis, and whether apoplexy at the end of a life is to be treated as an indication of insane heredity. Apoplexy is a generic term used for several pathological conditions: Trousseau enumerates five.

Brachet points out that the heredity of Isabella of Bourbon, the wife of Charles the Bold, has been overlooked. We have to go back six generations to find for this princess an insane ancestor, and the insanity was traumatic. On the third ascending generation a collateral, Jeanne de Bourbon, insane for a year, was mother of the mad king Charles VI. Isabella herself was never deranged, though her brother had violent headaches. She was the great grandmother of the Emperor Charles through Mary of Burgundy and the Archduke Philip, the husband of Juana, *la loca*.

Brachet denounces the sterility of several monographs by

physicians upon Joan of Arc for no better reason than that we have no details of the health histories of her family. He considers the accounts which Suetonius and Tacitus have given of the Cæsars as unworthy of attention because we cannot get at the sources from which these historians drew. All this betrays a jealousy of nimbler competitors who stepped before M. Brachet while he was toiling at his task of twenty years.

Brachet does not spare his own countrymen — Michelet, Sismondi, Monod, Martin and Moreau de Tours come in for hard knocks. He is suspicious even of the reports of the physicians who attend the great personages about whose diseases he writes, since the doctors only reproduced their own erroneous theories; yet it is likely that the physicians of the times would observe and describe symptoms better than the chroniclers or letter-writers, who only knew about the sick beds of the great through report or less distant, perhaps starting from the physicians he discredits.

Although we cannot endorse M. Brachet's claim to have been the pioneer "Bahnbrecher" in this field of historical research, he has a right to the credit of having thoroughly made his own the large territory which he has covered. To pursue this line of research upon his own method, he tells us, will be the work of the twentieth century.

M. Brachet observes at the end of his introduction: "People in a hurry will restrict themselves to reading my book; the true savant will pore over the proofs to seek new interpretations." One might naturally ask what interpretation does the learned author give of this mass of medical texts verified by severe methods of historical criticism? Will they reveal the laws of heredity? His answer is: "We do not know." Thus M. Brachet was content to collect the facts, leaving to others to interpret them. Perhaps if he had finished the introduction it might have contained the conclusions which he had approached. We doubt whether his book will find many readers, though we hope that it will not be neglected. The best service that could be done to M. Brachet's memory would be to peruse his book with a view to extract in a condensed form the lessons which may be deduced from it.

WILLIAM W. IRELAND.

La nature syphilitique et la curabilité du tabes et de la paralysie générale. Par L. E. LEREDDE. (pp. 141. Price 3.5 francs. C. Naud, Paris, 1903.)

BEFORE commencing the systematic portion of the book, the author presents a succinct account of his conclusions, which in brief are that tabes and general paralysis (*chez les syphilitiques*) are curable by mercurial treatment. He urges that hitherto mercurial treatment has not been systematic enough, and that the drug has been given in inadequate doses.

The title of the work is somewhat misleading, and in Chapter V. the author defines the meaning he attaches to the word cure, stating that the arrest of the progress of tabes is a cure, in the same sense that the cicatrisation of a cutaneous gumma is a cure.

But inasmuch as the changes in tabes cannot be termed gummatous the comparison is hardly justifiable, and inasmuch as the word tabes connotes a whole series of pathological changes with resultant symptoms and physical signs, the mere arrest can by no argument be termed a cure. The substitution of the word arrest for curability in the title would have avoided misconception and removed a certain amount of prejudice.

The first chapter is devoted to a brief discussion of the syphilitic origin of tabes and general paralysis, a proposition with which the author agrees, though reserving the opinion that such origin is not necessarily constant.

In Chapter II. a brief *résumé* of Professor Fournier's views on parasyphilis is given, and his view as to the incurability of such conditions is called in question.

Chapter III. is entitled "Incertitudes et Contradictions." Under "Guérison d'un cas de tabes" the author gives an account of a case in which cure resulted as the result of mercurial treatment. Unfortunately, though of little importance in view of the cases brought forward in Chapter V., the diagnosis of tabes in this particular case seems scarcely justified.

In the next section Professor J. Renaut gives a short account of the pathological anatomy of syphilis, and applies his conclusions to parasyphilitic affections, attributing practically all syphilitic processes to the cutting off of the blood supply to the area in question by the narrowing of the vessels, produced by endarteritis. Finally, in mentioning the scleroses produced in the central nervous system by syphilis, he concludes by remarking that "Rien n'autorise à dire que cette sclérose, une

fois formée, devient indépendante de la syphilis et demeure dans tous les cas irréductible,"—a statement in direct opposition to current views.

It can only be inferred from his remarks that in tabes the system degenerations are produced by the cutting off of the blood supply, say of the posterior roots. Now Mott,[1] as the result of his examinations of the posterior degenerated roots, states: "The walls of the arteries and arterioles were more often normal than not; when thickened, it was generally in a subject over fifty, and the thickening was not limited to the posterior roots, but affected also the arteries of the comparatively healthy anterior roots. I do not remember seeing one which presented the typical character of recent syphilitic arteritis, though it is not uncommon to find a condition of arterio-sclerosis, which might, of course, be indirectly of syphilitic origin."

Chapter V. contains a series of illustrative cases as reported by other observers, but singularly enough, none of M. Leredde's own. Of these cases, it may at once be said that many symptoms of undoubted tabes, under treatment by mercurial inunction, or hypodermic injections of mercury, either completely disappeared, or showed very considerable amelioration. It is impossible to go into any details, but the cases as recorded show striking improvement in such symptoms as lightning pains, bladder weakness, incoördination, &c. Similar improvement is claimed, and illustrative cases given, for general paralysis.

Finally, a chapter is devoted to the author's actual method of treatment. He advises that whatever salt be used, it should be administered in gradually increasing doses, so as ultimately to attain to $3\frac{1}{2}$ centigrammes (just over 5 grs.) of mercury daily. The injections are made either into the buttock or into the lumbar muscles. Preference is given to the soluble salts, such as the benzoate, rather than, for instance, to calomel made up with oil. Iodides he regards as useless for the sclerotic nervous changes.

Although the results claimed are very striking, it must be borne in mind that tabes is by no means always a progressive disease, and that in any large series a good many cases of arrest and of improvement will be seen apart from any mercurial treatment. The author realises the importance of early treatment, his view being presumably that then the lesion is a definitely syphilitic affection of the vessels of the posterior roots amenable to mercurial treatment. As indicated above, such arterial condition is not demonstrable. And it must be acknowledged that even apart

[1] *Archives of Neurology*, vol. ii., p. 264.

from treatment it is in the early stages of tabes that the prognosis is the most hopeful. Gowers states: "Only when the symptoms have developed rapidly and the syphilis is recent is there any possibility of benefit from anti-syphilitic treatment." M. Leredde would appear to adopt his method for advanced cases in spite of the general experience that in such cases mercurial treatment appears not infrequently to definitely hasten the progress of the disease. It is unfortunate, too, that in a point of such importance the author relies almost solely on cases reported by other observers. However, we think the work is of service in that it confirms the fact that tabes of recent onset has a better prognosis than in the later stages, that it confirms the benefit of mercurial treatment in certain cases, and that in those recorded striking improvement followed the use of active mercurial treatment.

ALFRED E. RUSSELL.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

At a Pathological Meeting held at 11, Chandos Street, on Thursday, December 3, at 8.30 p.m., the following communications were made :—

Dr. Farquhar Buzzard.

(1) Microscopic and lantern slides illustrating the descending spinal fibres of a lumbar posterior root in a human subject. The case on which the observations are based was that of a patient who died from malignant disease of the vertebral column and in whom the third lumbar posterior root on one side was compressed by the growth. The specimens show the results of examining the lumbar and sacral parts of the spinal cord by Busch's method.

(2) A dermoid tumour of the brain situated on the orbital surface of the frontal lobes in a boy of 14 years of age. Symptoms arising from the presence of the tumour had been present for about eighteen months before death.

Dr. Michell Clarke.

(1) Diphtheritic paralysis.—Microscopic specimens of slides showing extensive affection of cells in anterior horn, the anterior mesial group excepted, in the cervical and lumbar regions of cord, especially in the former, with chromatolytic changes, some cells in Clarke's column similarly affected; showing also degenerated fibres passing out from anterior horn into anterior roots, and degenerated fibres in hypoglossal and vagus roots, from a girl aged 20; paralysis affected the arms first, a week later the legs; extensor muscles predominantly affected; death in six weeks from paralysis of diaphragm.

(2) Microscopic specimen of gumma of cord in lumbar region; gumma is on left side of cord growing into it from periphery of lateral column. Atrophy and fibrillary tremor of muscles of left thigh and calf, with knee and ankle clonus, girdle pain on left side in region of tenth to twelfth dorsal nerves; no loss of

sensation. Patient, a man aged 48, died later in a series of fits, followed by right hemiplegia due to large gumma in cortex of left cerebral hemisphere.

(3) Microscopic specimen of internal pachymeningitis of cord in *hereditary syphilis* in a man aged 21. The meningeal thickening was most marked in upper dorsal region, where it produced myelitis from pressure, and lumbar regions, although it extended more or less along the whole cord from the lower cervical region downwards.

Dr. H. A. Caley.—*Hydatid of the brain-cyst wall; sections of cyst wall, scolices from the fluid.*

Specimens were obtained from a girl aged 14, who came under observation owing to ataxy of the cerebellar type, with commencing double optic neuritis more marked on the left side.

A few days later, owing to the evidently rapid increase of the intracranial pressure, it was decided to trephine. The left lobe of the cerebellum was first explored, with negative result, and with a view to relieving the tension it was then decided to trephine over the left lateral ventricle. A few ounces of clear fluid were withdrawn, which was subsequently found to contain numerous scolices.

Two days later free drainage was established, and a considerable quantity of fluid evacuated.

A few days later the cyst wall came out at the trephine opening during the dressing of the wound.

Dr. J. S. Collier.

(1) Fibres of the pyramidal tract ending in Clarke's column.
(2) Ending of the fibres of the sacral triangle.
(3) Specimens from cases of cerebral diplegia.

Dr. Wilfred Harris.—*Prefixed and postfixed types of brachial plexus.*

Herringham first drew attention to the fact that, though the origin of a given nerve-bundle may vary in its root origin yet it does not vary in its relative root supply to the other branches of the plexus. The whole plexus, in different individuals, may therefore be shifted up or down, and may be spoken of as prefixed or postfixed in either case. In a series of twenty-seven dissections of the brachial plexus, I have found an outer head of the ulnar present in 85 per cent., sometimes as a fine recurrent

branch, sometimes as a thick strand from the seventh root, or outer cord. In two of the cases, one with a very fine branch from the outer cord to the ulnar, and in one in which it was absent, I found a definite branch from the inner cord to join the nerve to the coraco-brachialis from the seventh root, and in another postfixed case a branch given off from the internal anterior thoracic to join the musculo-cutaneous. In seven out of nine cases examined I have found a well-marked branch from the first dorsal root to the posterior cord, entering the musculo-spiral nerve, although it is definitely stated by Herringham and most of the text-books that the first dorsal gives no posterior branch. This branch is larger in the postfixed cases, and its fibres probably reach the extensor communis, minimi digiti and indicis, and the extensors of the thumb. In such postfixed cases there is frequently a small ascending branch from the second dorsal to join the plexus. In such a postfixed plexus the branch from the seventh root to the ulnar will be small, or absent, and the nerve to the coraco-brachialis may be partially supplied by the eighth root. In such a case, too, the fifth root is likely to be small, perhaps less than half the size of the sixth root, with no descending branch from the fourth. The suprascapular will then get a definite bundle from the sixth root, instead of being supplied from the fourth and fifth, and the deltoid possibly, and the biceps certainly, will then get some motor fibres from the sixth root. In a prefixed plexus, however, the fifth root will be large, with probably a large descending branch from the fourth, which will divide into anterior and posterior divisions like all the other roots of the plexus. The suprascapular will get no fibres from the sixth root, and the deltoid, biceps, spinati, &c., the group of muscles familiar as suffering together in Erb's paralysis, will receive their entire motor supply from the fourth and fifth. Though the circumflex and musculo-cutaneous receive in these cases about half their fibres from the sixth root, I suggest that the fibres from the lower root are sensory mainly to the muscles, joints, skin, &c. This arrangement would be somewhat analogous to that Sherrington showed to exist for the vastus internus, in which the third lumbar is the efferent, and the fourth lumbar the afferent root for the reflex of the knee jerk. In the prefixed plexus, again, the nerve to the coraco-brachialis will arise equally from the sixth and seventh; the outer head of the ulnar will be large; the branch to the posterior cord from the first dorsal will be very small or absent, and the ascending branch from the second dorsal will also probably be absent. The whole plexus

may be, therefore, shifted up or down as much as nearly half a root, and this anatomical variation is of great importance in the diagnosis of nerve-root lesions. Thus, for example, a lesion of the fifth root in Erb's paralysis may in some cases, those of prefixed plexus probably, paralyse the radial extensors of the wrist, the pronator radii teres, and both the supinators completely, in addition to the deltoid spinati, biceps, and brachialis anticus; while in cases of postfixation of the plexus these peripheral muscles might escape partially or completely, and even the biceps and brachialis anticus would partially escape.

Dr. JAMES F. FLASHMAN.

A demonstration showing certain advantages of the clove-oil-celloidin embedding method, together with some remarks regarding a few improvements in the technique.

Dr. H. MORLEY FLETCHER and Dr. F. E. BATTEN.—*The pathology of a case of progressive muscular atrophy in a child aged* 11.

The disease ran its course in about eighteen months, starting with wasting and weakness of the hand, the legs being but little affected. There was also atrophy of the muscles of the tongue, weakness of muscles of the neck, weakness of lower intercostal and abdominal muscles. Sudden death occurred from respiratory failure.

The changes found on pathological examination were as follows :—

Extensive destruction and atrophy of the cells of the anterior horns from the upper cervical to the lower thoracic segments.

Extensive degeneration of the whole of the ventral region of the cord in the thoracic segments, the lesion being most marked at the level of the sixth thoracic segment. Degeneration in the direct cerebellar tracts. There was marked increase of the connective tissue in the ventral portion of the cord in the thoracic region.

The lumbar region of the cord showed but little change.

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EDITOR.

INDEX

TO

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FOR

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INDEX OF AUTHORS.

PAGE

Ascherson, W. L.
Case of idiopathic muscular atrophy (facio-scapulo-humeral type) 141

Batten, F. E.
Cases of myopathy, infantile type 147, 148
Myopathy, pseudo-hypertrophic type 146
On the diagnostic value of the position of the head in cases of cerebellar disease 71
Two cases of muscular atrophy of uncertain origin 151
Typical case of peroneal muscular atrophy 149

Batten, F. E., and Fletcher, H. Morley.
Case of progressive muscular atrophy of spinal origin in a girl aged 11 years; with autopsy 473, 615

Beyerman, D. H., and Langelaan, J. W.
On the localisation of a respiratory and a cardio-motor centre on the cortex of the frontal lobe... 81

Bishofswerder, L.
Contribution à l'étude de l'anatomie pathologique de la syringomyélie (Review) 139

Bolton, Charles.
Pathological changes in the medulla oblongata in acute diphtheritic toxæmia (Review) 283

Bolton, J. S.
The functions of the frontal lobes 215
The histological basis of amentia and dementia (Review)... ... 277

PAGE

Brachet, Auguste.
Pathologie mentale des rois de France (Review) 605

Bramwell, Byrom.
Case of hæmorrhage into central region of spinal cord 301
Case of hæmorrhage into latent syringomyelia 301
Case of removal of cerebral tumour 300
Case of tabes with optic atrophy 300
Case of tabes without ataxia 301

Bramwell, J. Milne.
Hypnotism, its history, practice and theory (Review) 602

Broadbent, Walter.
Cerebral embolus during an epileptic fit 447

Broadbent, W. H.
Hughlings Jackson as pioneer in nervous physiology and pathology 305

Bruce, Alexander.
Case of paralysis of serratus magnus 304
Localisation of function in the lumbo-sacral part of the cord ... 298

Buzzard, E. Farquhar.
Dermoid tumour of brain 612
Descending spinal fibres of a lumbar posterior root 612
On the pathology and bacteriology of Landry's paralysis 94
Progressive muscular atrophy in a young woman 472

Buzzard, E. Farquhar, and Collier, James S.
The degenerations resulting from lesions of posterior nerve roots and from transverse lesions of the spinal cord in man. A study of twenty cases 559

Buzzard, Thomas.
Case illustrating an ileo-psoas type of progressive muscular dystrophy 149

Caley, H. A.
Hydatid of a brain-cyst wall: sections of cyst wall; scolices from fluid 613

Clarke, J. Michell.
A case of Erb's juvenile dystrophy associated with bilateral enlargement of the parotid and submaxillary glands 202
Case of juvenile type of muscular atrophy with simultaneous enlargement of parotid glands 143
Diphtheritic paralysis... 612
Gumma of cord in lumbar region 612
Internal pachymeningitis of cord in heredita y syphilis 613

Clouston, T. S.
Cases of exophthalmic goitre with mental disease 300

Collier, James S.
A case of (?) disseminate sclerosis 471
Two cases of peroneal atrophy 149

PAGE

Collier, James S., and Buzzard, E. Farquhar.
The degenerations resulting from lesions of posterior nerve-roots and from transverse lesions of the spinal cord in man. A study of twenty cases 559

Cunningham, D. J.
Text-book of anatomy (Review) 461

Dean, G., and Usher, C. H.
Experimental research on the course of the optic fibres 524

Feindel, E., and Meige, H.
Les tics et leur traitement (Review) 462

Ferrand, Jean.
Essai sur l'hémiplégie des vieillards (Review) 459

Flatau, E.
Handbuch der pathologischen Anatomie des Nervensystems (Review) 285, 595

Fleming, R. A.
Case of cerebral compression 303

Fletcher, H. Morley, and Batten, F. E.
Case of progressive muscular atrophy of spinal origin in a girl aged 11 years; with autopsy 473, 615

Fletcher, W. M.
Tetanus dolorosus, and the relation of tetanus toxin to the sensory nerves and the spinal ganglia 383

Foerster, Otfrid.
Die Physiologie und Pathologie der Coördination, eine Analyse der Bewegungsstörungen bei der Erkrankungen des Centralnervensystems und ihre rationelle Therapie (Review)... 138

Gardner, James, and Thorburn, William.
A case of tumour of the axis, illustrating the functions of the third spinal segment 120

Gibson, G. A.
Case of right homonymous hemianopia 302

Gordon, Holmes.
Three cases of unilateral tremor in cerebral lesions 470

Grant, Dundas.
Case of hemianæsthesia affecting the skin and special senses with crossed amblyopia and achromatopsia 469
Case of paralysis of the palate and larynx 468
Case of paralysis of vocal cord with signs of involvement of the sympathetic nerve 470

PAGE

Greenfield, W. S.
Case of acromegaly 303

Halliburton, W. D., and Mott, F. W.
The coagulation temperature of cell globulin and its bearing on hyperpyrexia (Review) 281

Harris, Wilfred.
Post-diphtheritic chronic bulbar paralysis, and its distinction from myasthenia (with bibliography) 543
Prefixed and postfixed type of brachial plexus 613

Harris, Wilfred, and Low, V. Warren.
Case of Erb-Duchenne paralysis treated by operation 465
Case of infantile paralysis of the shoulder treated by operation ... 466

Head, Henry.
A case of pseudo-hypertrophic paralysis in an adult 146

Hepburn, D., and Waterston, D.
Shape of gray matter in spinal cord of porpoise 299

Horsley, Victor.
Three cases of cerebellar tumour after operation with reference to the question of cerebellar compensation 467

Hutchison, R.
Case of myopathy, Erb's juvenile type 146

Jackson, J. Hughlings.
Bibliography... 356
On the study of diseases of the nervous system 367

Janet, Pierre.
Les obsessions et la psychasthénie (Review) 449

Langelaan, J. W., and Beyerman, D. H.
On the localisation of a respiratory and a cardiomotor centre on the cortex of the frontal lobe 81

Langley, J. N.
The autonomic nervous system 1

Leredde, L. E.
La nature syphilitique et la curabilité du tabes et de la paralysie générale (Review) 609

Low, V. Warren, and Harris, Wilfred.
Case of Erb-Duchenne paralysis, treated by operation 465
Case of infantile paralysis of the shoulder, treated by operation ... 466

Lundie, R. A.
Case of infantile oculo-facial palsy 302

PAGE

McDougall, W.
The nature of inhibitory processes within the nervous system ... 153

Meige, H., and Feindel, E.
Les tics et leur traitement (Review) 462

Mott, F. W.
Archives of neurology from the pathological laboratory of the London County Asylums, Claybury, Essex (Review) 268
Note upon the choline test for active degeneration of the nervous system (Review) 284
Tabes in hospital and asylum practice (Review) 268
The prevention of dysentery in the London County Asylums (Review) 282

Mott, F. W., and Halliburton, W. D.
The coagulation-temperature of cell-globulin and its bearing on hyperpyrexia (Review) 281

Oppenheim, H.
Die Geschwülste des Gehirns (Review) 592

Ormerod, J. A.
Case of myopathy, Erb's juvenile type 144

Pick, A.
Clinical studies: (1) On "dreamy mental states" as a permanent condition in epileptics. (2) On the pathologically protracted duration of impressions on the senses as a cause of various disturbances of the sensory perception and especially of the sight. (3) On reduplicative paramnesia 242

Robertson, W. Ford.
Bacteriological, histological, and experimental investigations into the pathology of general paralysis 298

Russell, J. S. Risien.
Case of myopathy (idiopathic muscular atrophy), coming on late in life (facio-scapulo humeral type) 143

Smith, W. C.
The range of immediate association and memory in normal and pathological individuals (Review) 282

Spiller, Gustav.
The mind of man; a text-book of psychology (Review) 288

Spiller, W. G.
A report of two cases of paraplegia occurring in variola, one being a case of anterior poliomyelitis in an adult 134

Stewart, T. Grainger.
A case of diplegia and myoclonus 466

Stoddart, W. H. B.
The evolution of consciousness... 432

Sutherland, G. A.
Chorea and Graves' disease 210

PAGE

Taylor, James.
Case of myopathy with muscular twitching 151

Thomson, H. Campbell.
Associated movements in hemiplegia; their origin and physiological significance 514
Hereditary spastic paraplegia. Its relation to Friedreich's disease and its claim to be considered as a clinical entity 412

Thomson, John
Case of cerebellar disease 302
Case of fine general tremor 302

Thorburn, William, and Gardner, James.
A case of tumour of the axis, illustrating the functions of the third spinal segment 120

Tredgold, A. F.
Amentia (idiocy and imbecility) (Review) 274

Turner, John.
An account of the nerve cells in thirty-three cases of insanity, with special reference to those of the spinal ganglia 27
Notes on the minute structure of the human caudate nucleus and optic thalamus 400

Usher, C. H., and Dean, G.
Experimental research on the course of the optic fibres 524

Vires, J.
Maladies nerveuses, diagnostic, traitement (Review) 291

Waterston, D., and Hepburn, D.
Shape of gray matter in spinal cord of porpoise 299

Watson, Chalmers.
On "stringhalt" and "shivering" in horses. A study in comparative neuro-pathology 192

Watson, G. A.
The pathology and morbid histology of juvenile general paralysis (Review) 279

White, W. Charles.
Lesion of the brachium pontis with division of the trigeminal and facial nerves 440

Williams, Leonard.
A case of infantile hemiplegia of the right side, with paralysis agitans beginning on the left side 467

Williamson, R. T.
Note on "toxic" degeneration of the lower neurones simulating peripheral neuritis 206

Wright, Hamilton.
Beri-beri in monkeys 488

INDEX OF SUBJECTS.

PAGE

Acromegaly, case of. W. S. Greenfield 303

Amentia (idiocy and imbecility). A. F. Tredgold (Review) 274
and dementia, the histological basis of. J. S. Bolton (Review) ... 277

Anatomy, pathological, of nervous system, handbook of. E. Flatau, L. Jacobson and L. Minor (Review). 285, 595
text-book of. D. J. Cunningham (Review) 461

Association, range of immediate, and memory in normal and pathological individuals. W. C. Smith (Review) 282

Autonomic nervous system. J. N. Langley 1

Axis, tumour of, illustrating functions of third spinal segment. W. Thorburn and J. Gardner 120

Bacillus, resembling Klebs-Löffler bacillus in general paralysis. W. Ford Robertson 298

Bacteriology and pathology of Landry's paralysis 94

Beri-Beri in monkeys. Hamilton Wright 488

Brachium pontis, lesion of, with division of the trigeminal and facial nerves. W. C. White 440

Brachial plexus, prefixed and postfixed, types of. Wilfred Harris ... 613

Brain, dermoid tumour of. E. F. Buzzard 612
tumours of. H. Oppenheim (Review) 592

Bulbar autonomic system. J. N. Langley 2
paralysis, post-diphtheritic chronic, and its distinction from myasthenia (with bibliography). Wilfred Harris 543

Cardio-motor and respiratory centres, localisation of, on frontal cortex. J. W. Langelaan and D. H. Beyerman 81

Caudate nucleus and optic thalamus, minute structure of. John Turner 400

Cell-globulin, coagulation temperature of, and its bearing on hyperpyrexia. W. D. Halliburton and F. W. Mott (Review) ... 281

Cerebellar disease, diagnostic value of position of head in. F. E. Batten 71
disease, case of. John Thomson 302
tumour, three cases of, after operation, with reference to the question of cerebellar compensation. Victor Horsley 467

Cerebral compression, case of. R. A. Fleming 303
embolus during an epileptic fit. Walter Broadbent 447
tumour, case of removal of. Byrom Bramwell 300

Choline test for active degeneration of nervous system. F. W. Mott (Review) 284

Chorea and Graves' disease. G. A. Sutherland 210

Consciousness, the evolution of. W. H. B. Stoddart 432

Convulsions, Hughlings Jackson on. W. H. Broadbent 310

Coördination, Physiologie und Therapie der. O. Foerster (Review) ... 138

PAGE

Cortex, frontal, localisation of respiratory and cardiomotor centres on. J. W. Langelaan and D. H. Beyerman 81

Degeneration, active, of nervous system, choline test for. F. W. Mott (Review) 284
toxic, of lower neurones simulating peripheral neuritis. R. T. Williamson 206

Degenerations from lesions of posterior nerve roots, and from transverse lesions of spinal cord in man. J. S. Collier and E. F. Buzzard 559

Dementia and amentia, the histological basis of. J. S. Bolton (Review) 277

Diphtheritic toxæmia, changes in medulla oblongata in acute. Charles Bolton (Review) 283

Diplegia and myoclonus, case of. T. Grainger Stewart 466

Disseminated sclerosis, case of. J. S. Collier 471

Dissolution of the nervous system, factors of insanities in. W. H. Broadbent 329

Dreamy mental states as a permanent condition in epileptics. A. Pick 242

Dysentery, prevention of, in London County Asylums. F. W. Mott (Review) 282

Dystrophy, Erb's juvenile, with bilateral enlargement of the parotid and submaxillary glands. J. Michell Clarke... 202

Embolus, cerebral, during an epileptic fit. Walter Broadbent ... 447

Epilepsy, Hughlings Jackson on. W. H. Broadbent 312
pathology of, Hughlings Jackson's views on. W. H. Broadbent ... 317

Epileptic fit, cerebral embolus during an. Walter Broadbent ... 447

Epileptics, dreamy mental states as a permanent condition in. A. Pick 242

Erb-Duchenne paralysis, case of, treated by operation. W. Harris and V. W. Low 465

Evolution and dissolution, Hughlings Jackson on. W. H. Broadbent... 318

Exophthalmic goitre and mental disease, cases of. T. S. Clouston ... 300

Friedreich's disease, relation to hereditary spastic paraplegia. H. Campbell Thomson 412

Frontal cortex, localisation of respiratory and cardio-motor centres on. J. W. Langelaan and D. H. Beyerman 81

Frontal lobes, the functions of the. J. S. Bolton 215

Ganglia, spinal, cells of, in thirty-three cases of insanity. J. Turner 27

General paralysis, bacillus resembling Klebs-Löffler bacillus in. W. Ford Robertson 298
juvenile, pathology and morbid histology of. G. A. Watson (Review) 279
nature syphilitique et curabilité. L. E. Leredde (Review) ... 609

Geschwülste des Gehirns. H. Oppenheim (Review)... 592

Graves' disease and chorea. G. A. Sutherland 210

Hæmorrhage into central region of spinal cord, case of. Byrom Bramwell... 301
into latent syringomyelia. Byrom Bramwell 301

PAGE

Head, position of, in cerebellar disease, diagnostic value of. F. E. Batten 71

Hemianæthesia affecting skin and special senses, with crossed amblyopia and achromatopsia, case of. Dundas Grant 469

Hemianopia homonymous, case of right. G. A. Gibson 302

Hemiplegia, associated movements in, their origin and physiological significance. H. Campbell Thomson 514
infantile, of right side, with paralysis agitans on left side. Leonard Williams 467

Hémiplégie des vieillards, essai sur l'. J. Ferrand (Review) 459

Hereditary spastic paraplegia, relation to Friedreich's disease. H. Campbell Thomson 412

Hydatid of brain. H. A. Caley 613

Hyperpyrexia, bearing of coagulation temperature of cell-globulin on. W. D. Halliburton and F. W. Mott (Review)... 281

Hypnotism, history, practice and theory. J. Milne Bramwell (Review) 602

Infantile paralysis of the shoulder treated by operation. W. Harris and V. W. Low 466

Inhibitory processes within the nervous system, the nature of. W. MacDougall 153

Insanity, nerve cells in thirty-three cases of, with special reference to the spinal ganglia. J. Turner 27

Insanities, factors of, in dissolution of the nervous system. W. H. Broadbent 329

Jackson, Hughlings, as pioneer in nervous physiology and pathology. W. H. Broadbent 305

Landry's paralysis, pathology and bacteriology of. E. F. Buzzard ... 94
bibliography 118

Language, Hughlings Jackson's analysis of. W. H. Broadbent ... 345

Larynx, paralysis of palate and. Dundas Grant 468

Lobes, frontal, the functions of the. J. S. Bolton 215

Localisation of a respiratory and a cardio-motor centre on the frontal cortex. J. W. Langelaan and D. H. Beyerman 81
of function in lumbo-sacral part of spinal cord. Alexander Bruce ... 298

Medulla oblongata, pathological changes in, in acute diphtheritic toxæmia. Charles Bolton (Review) 283

Memory, range of immediate association and, in normal and pathological individuals. W. C. Smith (Review) 282

Mental disease, exophthalmic goitre and, cases of. T. S. Clouston ... 300
pathology of the Kings of France. Aug. Brachet (Review) ... 605

Micrococcus thecalis in Landry's paralysis. E. F. Buzzard 114

Mid-brain autonomic system. J. N. Langley 2

Mind of man. A text-book of psychology. G. Spiller (Review) ... 288

Movements, associated, in hemiplegia. H. Campbell Thomson ... 514

PAGE

Muscular atrophy, progressive, in a young woman. E. Farquhar Buzzard 472
progressive, of spinal origin; with autopsy. H. Morley Fletcher and F. E. Batten 473, 615

Myasthenia, distinction from post-diphtheritic bulbar paralysis. Wilfred Harris 543

Myoclonus and diplegia, case of. T. Grainger Stewart 466

Myopathy, case of, with muscular twitching. James Taylor ... 151
Erb's juvenile dystrophy with bilateral enlargement of parotid and submaxillary glands. J. Michell Clarke 202
facio-scapulo-humeral type (Landouzy-Dejerine), case of. W. L. Ascherson 141
case of, late in life. J. S. R. Russell 143
ileo-psoas type, case of. Thomas Buzzard 149
infantile type, three cases of. F. E. Batten 147
cases of. F. E. Batten 147, 148
juvenile type (Erb), case of. J. Michell Clarke 143
case of. J. A. Ormerod 144
case of. R. Hutchison 146
peroneal type, two cases of. J. S. Collier 149
case of. F. E. Batten 149
pseudo-hypertrophic type, case of. F. E. Batten 146
case of. H. Head 146
two cases of muscular atrophy of uncertain origin. F. E. Batten... 151

Nerve-cells, an account of the, in thirty-three cases of insanity, with special reference to those of the spinal ganglia. J. Turner ... 27

Nerve-roots, posterior, degenerations from lesions of. J. S. Collier and E. F. Buzzard 559

Nerves, trigeminal and facial, division of, with lesion of the brachium pontis. W. C. White 440

Nervous diseases, diagnosis and treatment. J. Vires (Review) ... 291
physiology and pathology, Hughlings Jackson as pioneer in. W. H. Broadbent 305

Nervous system, on the study of diseases of the. J. Hughlings Jackson 367
choline test for active degeneration of. F. W. Mott (Review) ... 284
inhibitory processes within the, nature of. W. McDougall ... 153
pathological anatomy of, handbook of. E. Flatau, L. Jacobson, and L. Minor (Review) 285, 595
the autonomic. J. N. Langley 1

Neuritis, peripheral, simulation of, by toxic degeneration of lower neurones. R. T. Williamson 206

Neurological Society, proceedings of 141, 152, 293, 465, 612

Neurology, Archives of. F. W. Mott (Review) 268

Neurones, lower, toxic degeneration of, simulating peripheral neuritis. R. T. Williamson 206

Nucleus, caudate, and optic thalamus, minute structure of. John Turner 400

Obsessions, les, et la psychasthénie. Pierre Janet (Review) 449

Oculo-facial palsy (Gowers) case of infantile. R. A. Lundie 302

PAGE

Optic atrophy, case of tabes with. Byrom Bramwell 300
fibres, experimental research on the course of. G. Dean and C. H. Usher 524
thalamus and caudate nucleus, minute structure of. John Turner... 400

Palate, paralysis of larynx and. Dundas Grant 468

Palsy, infantile oculo-facial (Gowers), case of. R. A. Lundie ... 302

Paralysis, agitans, left sided, with right infantile hemiplegia. Leonard Williams 467
diphtheritic. Michell Clarke 612
Erb-Duchenne, treated by operation. W. Harris and W. V. Low ... 465
infantile, of shoulder treated by operation. W. Harris and V. W. Low 466
Landry's, pathology and bacteriology of. E. F. Buzzard 94
of palate and larynx, case of. Dundas Grant 468
of serratus magnus, case of. Alexander Bruce 304
of vocal cord with involvement of sympathetic, case of. Dundas Grant 470
post-diphtheritic chronic bulbar, and its distinction from myasthenia. Wilfred Harris 543

Paramnesia, reduplicative. A. Pick 260

Paraplegia, hereditary spastic, its relation to Friedreich's disease. H. Campbell Thomson 412
two cases occurring in variola. W. G. Spiller. 424

Pathology, nervous, and physiology, Hughlings Jackson as pioneer in. W. H. Broadbent 305
of juvenile general paralysis. G. A. Watson (Review) 279
of Landry's paralysis and its bacteriology. E. F. Buzzard ... 94

Pathologie mentale des rois de France. Aug. Brachet (Review) ... 605

Perception, sensory, disturbances of, by pathologically protracted duration of impressions on the senses. A. Pick 251

Physiology, nervous, and pathology, Hughlings Jackson as pioneer in. W. H. Broadbent 305

Poliomyelitis, anterior, in an adult, occurring in variola. W. G. Spiller 424

Pons, lesion of brachium, with division of the trigeminal and facial nerves. W. C. White 440

Progressive muscular atrophy, in a young woman. E. Farquhar Buzzard 472
of spinal origin; with autopsy. H. Morley Fletcher and F. E. Batten 473, 615

Psychasthénie, les obsessions et la. Pierre Janet (Review) 449

Psychology, the mind of man, a text-book of. G. Spiller (Review) ... 288

Respiratory and cardio-motor centres, localisation of, on frontal cortex. J. W. Langelaan and D. H. Beyerman 81

Sacral autonomic system. J. N. Langley 3

Sclerosis disseminate, case of. J. S. Collier 471

Senses, disturbance of sensory perception resulting from pathologically protracted duration of impressions on the. A. Pick 251

Sensory nerves and spinal ganglia, tetanus dolorosus and the relation of the tetanus toxin to. W. M. Fletcher 383

PAGE

"Shivering" and "stringhalt" in horses. Chalmers Watson ... 192

Spastic paraplegia, hereditary, relation to Friedreich's disease. H. Campbell Thompson 412

Spinal cord, degenerations from transverse lesions of. J. S. Collier and E. F. Buzzard 559
gumma of, in lumbar region. Michell Clarke 612
hæmorrhage into central region of. Byrom Bramwell 301
localisation of function in lumbo-sacral part of. Alexander Bruce... 298
of porpoise, shape of gray matter in. D. Waterston and D. Hepburn 299
pachymeningitis, internal, in hereditary syphilis. Michell Clarke 613
third segment, functions of. W. Thorburn and J. Gardner ... 120

Spinal, descending, fibres of a lumbar posterior root. E. F. Buzzard ... 612
ganglia and sensory nerves, tetanus dolorosus and the relation of the tetanus toxin to. W. M. Fletcher 383
ganglia, cells of, in thirty-three cases of insanity. J. Turner ... 27

"Stringhalt" and "shivering" in horses. Chalmers Watson ... 192

Sympathetic system. J. N. Langley 2

Syphilis, hereditary, internal pachymeningitis of cord in. Michell Clarke 613

Syringomyelia, latent, hæmorrhage into. Byrom Bramwell... ... 301

Syringomyélie, anatomie pathologique de la. L. Bischofswerder (Review) 139

Tabes dorsalis, case of, with optic atrophy. Byrom Bramwell ... 300
in hospital and asylum practice. F. W. Mott (Review) 268
nature syphilitique et la curabilité du. L. E. Leredde (Review) .. 609
without ataxia, case of. Byrom Bramwell 301

Tetanus dolorosus and the relation of tetanus toxin to the sensory nerves and the spinal ganglia. W. M. Fletcher 383
bibliography 399

Third spinal segment, functions of. W. Thorburn and J. Gardner ... 120

Tics, les, et leur traitement. H. Meige and E. Feindel (Review) ... 462

Toxæmia, diphtheritic, changes in medulla oblongata in acute. Charles Bolton (Review) 283

Toxic degeneration of lower neurones simulating peripheral neuritis. R. T. Williamson 206

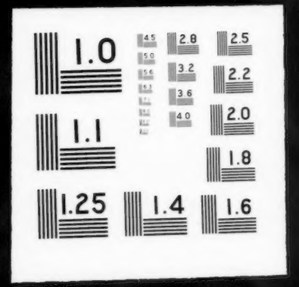
Toxin, tetanus, relation of, to sensory nerves and spinal ganglia. W. M. Fletcher 383

Tremor, case of fine general. John Thomson 302
unilateral, in cerebral lesions, three cases of. Gordon Holmes ... 470

Tumour, cerebellar, three cases of, after operation, with reference to the question of cerebellar compensation. Victor Horsley 467
cerebral, case of removal of. Byrom Bramwell 300
of axis, illustrating functions of third spinal segment. W. Thorburn and J. Gardner 120

Variola, two cases of paraplegia occurring in. W. G. Spiller 424

RESOLUTION CHART



100 MILLIMETERS

INSTRUCTIONS Resolution is expressed in terms of the lines per millimeter recorded by a particular film under specified conditions. Numerals in chart indicate the number of lines per millimeter in adjacent "T-shaped" groupings.

In microfilming, it is necessary to determine the reduction ratio and multiply the number of lines in the chart by this value to find the number of lines recorded by the film. As an aid in determining the reduction ratio, the line above is 100 millimeters in length. Measuring this line in the film image and dividing the length into 100 gives the reduction ratio. Example: the line is 20 mm. long in the film image, and $100/20 = 5$.

Examine "T-shaped" line groupings in the film with microscope, and note the number adjacent to finest lines recorded sharply and distinctly. Multiply this number by the reduction factor to obtain resolving power in lines per millimeter. Example: 7.9 group of lines is clearly recorded while lines in the 10.0 group are not distinctly separated. Reduction ratio is 5, and $7.9 \times 5 = 39.5$ lines per millimeter recorded satisfactorily. $10.0 \times 5 = 50$ lines per millimeter which are not recorded satisfactorily. Under the particular conditions, maximum resolution is between 39.5 and 50 lines per millimeter.

Resolution, as measured on the film, is a test of the entire photographic system, including lens, exposure, processing, and other factors. These rarely utilize maximum resolution of the film. Vibrations during exposure, lack of critical focus, and exposures yielding very dense negatives are to be avoided.